contaminates the data set by a purposeful action, such as blowing smoke into the inlet, or forgetting to wear the monitor and not admitting that error in the log of daily activity.

These unquantifiable "errors" in a PM PEM measurement study may be greater than the filter weighing errors and flow rate measurement errors that can be quality controlled through calibration procedures. This may be important for interpretation of published PM PEM data because these errors likely inflate the variance of the measurements.

7.3.2 Characterization of Particulate Matter Collected by Personal Monitors

The amount of PM collected by different types of personal monitors with the identical nominal cut-point can be variable. The difference between two PM measurements, made by two nominally identical monitors of different design, can be a function of the wind speed and the size distribution of the PM in the air mass being sampled. A recent field comparison by Groves et al. (1994) of different types of respirable dust samplers used in occupational settings where coarse mode PM predominates shows that there is considerable difference between the mass collected by sets of paired cyclones and paired impactors sampling in a concentration range of 500 to 6600 $\mu g/m^3$. The cyclones collected from 53 to 165% of the mass collected by the impactors. This type of comparison study has not been done for personal monitors used in nonoccupational studies at ambient and indoor respirable PM concentrations on the order of 10 to 100 $\mu g/m^3$, where the fine mode can be more important.

7.3.3 Microscale Variation and the Personal Cloud Effect

The study of Thatcher and Layton (1995) described in Section 7.2.2.2 reports the increase of indoor PM of various size ranges from household activities, such as walking into and out of a room. The tendency for such human activity in the home or at work to generate a "personal activity cloud" of particles from clothing and other items (stuffed furniture, carpet, etc.), that will be intense in the breathing zone and diluted near an area monitor located several meters away, has also been cited as a contributing factor to the discrepancy between personal measures of exposure and time-weighted-average (TWA) exposures using microenvironmental measurements (Martinelli et al., 1983; Cohen et al., 1984; Rodes et al., 1991). Fletcher and Johnson (1988) also measured metal concentrations (measurement method and size unspecified)

in an occupational exposure situation (metal spraying of spindles on a lathe) and found 50% higher concentrations measured from the left lapel compared to the right lapel, which reflected the orientation of the operator to the lathe.

7.4 NEW LITERATURE ON PARTICLE EXPOSURES SINCE 1981

The following sections review studies that measured PEM PM in the general non-smoking population. In these studies, the subjects spent time at home and in other indoor environments that include time at work. In the USA, recent data indicate that on a daily basis, an average US resident spends approximately 21 h indoors (85.6%), 100 minutes in (or near) a vehicle (7.2%), and 100 minutes outdoors (7.2%) (U.S. Environmental Protection Agency, 1989).

Almost all the studies of PM exposure in the general public have been conducted on urban and suburban residents. These subjects are often working in occupations that do not require PM monitoring to assure that occupational standards are being met (e.g. in an office). However, PM monitoring in an industrial workplace by a subject - independently of an official corporate industrial hygiene program - can have legal or security implications for an employer. A further complication arises from the fact that industrial exposures tend to be dominated by a specific type of particle. Coal miners are exposed to coal dust, textile workers are exposed to cotton dust, etc.

7.4.1 Personal Exposures in U.S. Studies

Dockery and Spengler (1981b) compared personal $PM_{3.5}$ exposures and ambient $PM_{3.5}$ concentrations in Watertown, MA, and in Steubenville, OH. In Watertown, 24-h personal samples were collected on a 1-in 6-day schedule, and in Steubenville, 12-h personal samples (8 a.m. to 8 p.m.) were collected on a Monday-Wednesday-Friday schedule. A correlation coefficient of 0.692 between the mean personal and the mean ambient concentration for 37 subjects, 18 in Watertown and 19 in Steubenville, was reported for the pooled data. However, this appears to be an artifact of two separate clusters formed by these data, each with considerably lower correlation. When these data are analyzed separately, the regression coefficient between personal and ambient for Watertown is $R^2 = 0.00$ and for Steubenville it is $R^2 = 0.18$.

Sexton et al. (1984) studied personal exposures to respirable particles ($PM_{3.5}$) for 48 nonsmokers during a winter period in Waterbury, VT, where firewood was either the primary or secondary heating source for the subject. Their results showed that personal exposures were 45% higher than indoor averages (36 $\mu g/m^3$ versus 25 $\mu g/m^3$) and indoor averages were 45% higher than outdoor averages (25 $\mu g/m^3$ versus 17 $\mu g/m^3$). Ambient air pollution, measured by an identical stationary ambient monitor (SAM) outside each residence (a pump contained in a heated box was connected to an external cyclone and filter), had no correlation with the residents' personal exposures ($R^2 = 0.00$) and 95% of the subjects had personal exposures *greater* than the median outdoor concentration.

Spengler et al. (1985) reported a study of $PM_{3.5}$ exposures in the non-industrial cities of Kingston and Harriman, TN, during the winter months of February through March, 1981. In this study, two Harvard/EPRI $PM_{3.5}$ monitors were used for each person. One stationary indoor monitor (SIM) remained indoors in the home, and the second monitor (PEM) was carried for 24-h to obtain the personal exposure. In each community, identical Harvard/EPRI samplers (SAM) were placed at a central site to represent ambient $PM_{3.5}$ concentrations. The results of the study are shown in Table 7-19. In both communities, 95% of the subjects had personal exposures to $PM_{3.5}$ *greater* than the average ambient concentrations. The mean personal exposure and indoor concentrations (44 ± 3 $\mu g/m^3$ and 42 ± 3 $\mu g/m^3$) were more than 100% greater than the mean ambient average of 18 ± 2 $\mu g/m^3$ sampled on the same days.

For the complete cohort, the correlation between PM PEM and PM SAM was $r = 0.07$ ($p = 0.30$), and between PM PEM and PM SIM was $r = 0.70$ ($p = 0.0001$). The correlation between simultaneous PM PEM and PM SAM was $r = 0.15$ for 162 nonsmoke exposed individual observations ($p = 0.06$). For 63 observations on smoke exposed individuals, the correlation $r = 0.16$ was not significant ($p = 0.16$) between PM PEM and PM SAM. An important finding was that in nonsmoking households, the PM PEM is always higher than SIM and SAM. "This implies that individuals encounter elevated concentrations away from home and/or that home concentrations are elevated while they are at home and reduced while they are away". This observation is supported by the findings of Thatcher and

TABLE 7-19. QUANTILE DESCRIPTION OF PERSONAL, INDOOR, AND OUTDOOR $PM_{3.5}$ CONCENTRATIONS ($\mu g/m^3$), BY LOCATION IN TWO TENNESSEE COMMUNITIES

City	Group	N	95%	75%	50%	25%	5%	Mean	S.E.
Kingston	Personal	133	99	47	34	26	19	42	2.5
	Indoor	138	110	47	31	20	10	42	3.5
	Outdoor	40	28	22	16	12	6	17	2.7
Harriman	Personal	93	122	54	35	24	15	47	4.8
	Indoor	106	129	45	27	18	10	42	4.1
	Outdoor	21	34	23	15	13	9	18	4.0
Total[a]	Personal	249	113	48	34	26	17	44	2.8
	Indoor	266	119	46	29	20	10	42	2.6
	Outdoor	71	33	23	17	13	7	18	2.1

[a]Includes samples from 13 subjects living outside Kingston and Harriman town limits and from four field personnel residing in these communities.
N = number of samples.
S.E. = Standard error.

Source: Spengler et al. (1985).

Layton (1995), reported in Section 7.2.2.2: merely walking into a room can raise the concentrations of PM by 100%. This study is relevant to the analyses by Dockery et al. (1992) of PM mortality in St. Louis, MO, and in Eastern Tennessee counties surrounding Kingston and Harriman as discussed in Chapter 12. Although the Spengler et al. (1985) and Dockery et al. (1992) studies are not directly comparable, because different years of data were used (1981 versus 1985/1986), the authors' assumption in Dockery et al. (1992) that the Harriman, TN, data represent exposures to PM in all of eastern Tennessee is called into question.

Morandi et al. (1988) investigated the relationship between personal exposures to PM and indoor and outdoor PM concentrations, using a TSI Model 3500 piezobalance that measures respirable particles in the range <3.5 μm. For the group of 30 asthmatics in Houston, TX, that were studied, outdoor concentrations averaged 22 $\mu g/m^3$, indoor concentrations averaged 22% higher than outdoor (27 $\mu g/m^3$) and, in motor vehicles, the average concentration of particles was 60% higher than the average outdoors (35$\mu g/m^3$). Personal 12-h (7 a.m. to 7 p.m.) daytime exposures to PM were not predicted as well by fixed site dichotomous sampler ambient monitors

(R^2 = 0.34) as by the indoor exposures (R^2 = 0.57). However, for 1-h exposures, they found no correlation (R^2 = 0.00) between the personal exposures to PM_5 and the indoor exposures measured with a TSI model 5000 stationary continuous piezobalance located in the "den" area of the home. The authors noted that use of home air conditioning and recirculation tended to increase the PM exposures.

Lioy et al. (1990) reported a study done during the winter (January 1988) in the industrial community of Phillipsburg, NJ, where personal PM_{10} was monitored along with indoor and outdoor PM_{10}. They collected PM_{10} (fine plus coarse particles on a single filter). In this study of eight residences of 14 nonsmoking individuals not smoke exposed at home, geometric mean 24-h concentrations were 68, 48 and 42 $\mu g/m^3$ for personal, outdoor and indoor sites, respectively. The arithmetic mean personal PM exposure of 86 $\mu g/m^3$ was 45% higher than the mean ambient concentration of 60 $\mu g/m^3$. The higher ambient than indoor concentrations in this study, a reversal of the relationships found in the Sexton et al. (1984), Spengler et al. (1985) and Morandi et al. (1988) studies, may be caused by the local industrial source of coarse particles in that community and the absence of cigarette smokers in the residences sampled. This difference also may be partially explained by the 10 µm particle sizes sampled in the NJ study and the 3.5 µm particle sizes in the other studies. The regression coefficient between personal and ambient PM_{10} for all 14 people on the 14 days of the study (n = 191 valid personal values) was 0.19 (R^2 = 0.037, p = 0.008). With three personal exposure extreme values removed (n = 188 personal values) and without correction for missing data, the coefficient was 0.50 (R^2 = 0.25, p = 0.007).

Lioy et al. (1990) report individual regression equations of PEM and SAM for the six of 14 subjects with significant relationships ($p < 0.01$). These data are shown in Table 7-20. For individuals with constant daily activities in the same microenvironments, the increment of PM exposure due to nonambient sources is repeatable with lower variability than that of the ambient PM. Therefore their variation of personal exposure from day-to-day is highly driven by the variation of the ambient PM. For subjects with intermittent exposures to nonambient PM, through non-repetitive activity patterns or intermittent source operation, the regression of PEM on SAM can become non-significant. This improvement in

TABLE 7-20. REGRESSION EQUATION OF THOSE INDIVIDUALS HAVING STATISTICALLY SIGNIFICANT RELATIONSHIPS OF EXPOSURE (PEM) WITH OUTDOOR AIR CONCENTRATIONS (SAM)

Participant	Equation	R^2	N	p
01	y = 0.62 (0.12) X + 26.5 (17.3)	0.66	14	< 0.01
31	y = 0.55 (0.07) X + 7.3 (9.9)	0.83	14	< 0.01
52	y = 0.63 (0.11) X + 15.3 (14.7)	0.74	14	< 0.01
62	y = 1.29 (0.27) X + 33.0 (37.1)	0.67	13	< 0.01
81	y = 1.07 (0.24) X + 39.0 (32.6)	0.63	14	< 0.01
91	y = 0.59 (0.12) X + 42.0 (19.9)	0.63	13	< 0.01

y = Personal air PM-10.
X = Outdoor air PM-10.
( ) = Confidence interval.

Source: Lioy et al. (1990).

correlation was also shown for their indoor versus outdoor relationships, between cross-sectional and individual comparisons, as described in Section 7.4.2.3.

In all these studies, the personal PM was measured to be higher than either the indoor or the outdoor PM measurements. This relationship of PEM > SIM and PEM > SAM has also been found in the PTEAM study (Clayton et al., 1993) described in detail in Section 7.2.2.1.3 and later in Section 7.4.1.1. For the PTEAM study during the day (7 a.m. to 7 p.m.) average personal PM_{10} exposure data (150 $\mu g/m^3$) were 57% higher than the average indoor and outdoor concentrations, which were virtually equal (95 $\mu g/m^3$). Consequently, a time-weighted-average (TWA) of the daytime indoor and outdoor PM concentrations appears to always underestimate the personal exposures to PM because the daytime PEM data are higher than either the SIM or SAM data. At night (7 p.m. to 7 a.m.) average PM_{10} personal exposures (77 $\mu g/m^3$) were higher than the average indoor concentrations (63 $\mu g/m^3$) but lower than the average outdoor concentration (86 $\mu g/m^3$).

It has been proposed (World Health Organization, 1982a; Spengler et al., 1985; Mage, 1985) that such a discrepancy between the TWA and the personal monitoring measurements may be caused by two factors described as follows: (1) human exposure to PM at work and in traffic are only partially accounted for in a TWA of indoor and outdoor ambient PM values; and (2)

indoor and outdoor averages reflect periods of low concentration during which the subject is not present. The PM pollution generating activities in a home usually occur only when a person is at home, as discussed in Section 7.1.2 concerning Equation 7-2. Therefore, the PM in a home will be higher when a person is present than when the home is unoccupied. A 24-h average of the indoor concentration thereby underestimates the average exposure of a person while in that home.

Ambient PM is also higher during the day (when industry and traffic are active, and wind speeds are high) than at night when PM generating activities are at a minimum and the air is still (Miller and Thompson, 1970). Consequently, a 24-h average ambient PM value generally underpredicts the concentrations during the daylight hours and the exposures of people going outdoors during that period.

7.4.1.1 The Particle Total Exposure Assessment Methodology Study

In 1986, the U. S. Congress mandated that EPA's Office of Research and Development "carry out a TEAM Study of human exposure to particles." The main goal of the study was to estimate the frequency distribution of exposures to particles for nonsmoking Riverside, CA, residents. Another goal was to determine particle concentrations in the participants' homes and immediately outside the homes. The detailed analyses of the indoor PM and outdoor PM data were described in Section 7.2.2.1.3.

7.4.1.1.1 Pilot Study

Study Design

A prepilot study, described in Section 7.2.2.1.3, was undertaken in nine homes in Azusa, CA in March of 1989 to test the sampling equipment (Özkaynak et al., 1990). Newly-designed personal exposure monitors (PEMs) were equipped with thoracic (PM_{10}) and fine ($PM_{2.5}$) particle inlets. The PEMs were impactors with 4-Lpm Casella pumps (Wiener, 1988). Two persons in each household wore the PEMs for two consecutive 12-h periods (night and day). Each day they alternated inlet nozzles. A central site with a PEM, a microenvironmental monitor (MEM), and two EPA reference methods (dichotomous and high-volume samplers) with a 10 μm size-selective inlet was also operated throughout the 11 days (22 12-h periods) of the study.

Results

The personal exposure levels were about twice as great as the indoor or outdoor concentrations for both PM_{10} (Table 7-21a) and $PM_{2.5}$ (Table 7-21b). Considerable effort was expended to demonstrate that this was not a sampling artifact, due for example to the constant motion of the sampler; however, no evidence could be found for an artifactual effect. Nonetheless, to reduce chances for an artifactual finding in the main study, it was decided to use identical PEMs for both the personal and fixed (indoor and outdoor) samples in the main study. Cross-sectional personal exposures were essentially uncorrelated (slightly negatively) with outdoor concentrations (R^2 = 0 to 2%) (Özkaynak et al., 1993a). However, a serial correlation analysis of these pilot PTEAM data were performed for the six or eight 12-h averages that comprised the three or four 24-h averages reported for the residents of the first five homes in Table 7-21a,b. The residents of four homes only carried the PEM for two days, so the four 12-h individual measurements were too few for development of a meaningful serial relationship. The results for the ten people in homes 1 to 5 are shown in Table 7-22. The medians of R^2 equal 0.12 for PEM $PM_{2.5}$ vs SAM $PM_{2.5}$ and 0.07 for PEM PM_{10} vs SAM PM_{10}, neither of which is significant. More importantly, the serial slopes were positive for 15 of the 20 cases which is the expected behavior, as opposed to the counter-intuitive negative correlation found for the pooled PEM vs SAM data for all residents of the nine homes.

In Azusa, the excess $PM_{2.5}$ and PM_{10} generated by personal activities increased the personal exposures by approximately 100% above the average of the indoor and outdoor values. These results are in marked contrast to the data of Tamura and Ando (1994) and Tamura et al. (1996) in which seven Japanese elderly housewives and male retirees had PM_{10} PEM exposures less than the time weighted average of SIM and SAM PM_{10} concentrations.

7.4.1.1.2 Main Study

Study Design

Ultimately 178 residents of Riverside, CA took part in the study in the fall of 1990. Respondents represented 139,000 ± 16,000 (S.E.) nonsmoking Riverside residents aged 10 and above. Their homes represented about 60,000 Riverside homes. Each participant wore the PEM for two consecutive 12-h periods. Concurrent PM_{10} and $PM_{2.5}$ samples were

TABLE 7-21a. PARTICLE TOTAL EXPOSURE ASSESSMENT METHODOLOGY PREPILOT STUDY: 24-HOUR PM_{10} CONCENTRATIONS ($\mu g/m^3$)

House	Day	Person 1	Person 2	Indoors	Outdoors
1	1	102	86	54	132
1	3	142	125	38	49
1	5	158	150	49	70
1	7	92	127	34	49
2	1	109	158	122	112
2	3	99	140	37	48
2	5	131	87	41	70
2	7	62	56	32	46
3	1	98	107	86	115
3	3	100	141	39	45
3	5	143	132	71	79
3	7	76	103	36	44
4	2	109	92	77	102
4	4	90	77	34	47
4	6	99	122	36	37
5	2	80	104	76	99
5	4	70	77	62	65
5	6	80	78	54	50
6	8	130	152	114	39
6	10	150	102	106	51
7	9	209	126	46	72
7	11	80	71	29	39
8	9	135	178	73	59
8	11	97	151	38	28
9	8	136	102	63	43
9	10	273	91	121	48
Mean		117.2	112.9	60.3	63.0
SD		44.9	30.8	28.5	27.1
SE		8.8	6.0	5.6	5.3

Source: Data from PTEAM Prepilot Study used to calculate R^2 values as shown in Table 7-22 and published by Wallace (1996).

TABLE 7-21b. PARTICLE TOTAL EXPOSURE ASSESSMENT METHODOLOGY PREPILOT STUDY: 24-H $PM_{2.5}$ CONCENTRATIONS ($\mu g/m^3$)

House	Day	Person 1	Person 2	Indoors	Outdoors
1	2	44	96	22	67
1	4	55	88	25	39
1	6	55	382	21	33
2	2	58	53	31	52
2	4	46	100	27	43
2	6	51	50	28	40
3	2	53	66	48	58
3	4	62	94	30	35
3	6	109	88	39	39
4	1	75	61	33	71
4	3	46	43	19	29
4	5	118	94	31	46
4	7	40	40	17	26
5	1	65	69	62	96
5	3	59	70	35	38
5	5	40	56	42	55
5	7	34	53	25	28
6	9	71	81	56	33
6	11	77	75	53	18
7	8	64	135	17	27
7	10	111	67	32	35
8	8	53	100	27	27
8	10	110	1453*	35	35
9	9	178	48	70	40
9	11	105	58	42	28
Mean		71.2	140.8*	34.7	41.6
SD		32.7	275.5	13.7	16.8
SE		6.5	55.1	2.7	3.4

* Horseback riding at an indoor ring. If this point is deleted, mean = 86.1.

Source: Data from PTEAM Prepilot Study used to calculate R^2 values as shown in Table 7-22 and published by Wallace (1996).

TABLE 7-22. REGRESSIONS OF PERSONAL EXPOSURE ON INDOOR AND OUTDOOR PM_{10} AND $PM_{2.5}$ CONCENTRATIONS: PARTICULE TOTAL EXPOSURE ASSESSMENT METHODOLOGY PREPILOT STUDY

House	Person	N	Intercept	SE	p	Slope	SE	p	R^2
PM_{10}: Personal vs. Outdoor									
1	1	8	124	42	0.03	−0.0004	0.51	NS	0
	2	8	134	60	NS	−0.16	0.73	NS	0.01
2	1	8	47	44	NS	0.77	0.58	NS	0.23
	2	8	26	52	NS	1.22	0.68	NS	0.35
3	1	8	83	47	NS	0.3	0.61	NS	0.04
	2	8	116	54	NS	0.07	0.7	NS	0.002
4	1	6	87	20	0.01	0.2	0.29	NS	0.1
	2	6	106	28	0.02	−0.15	0.4	NS	0.03
5	1	6	47	31	NS	0.42	0.41	NS	0.2
	2	6	22	26	NS	0.9	0.35	NS	0.63
$PM_{2.5}$: Personal vs. Outdoor									
1	1	6	41	20	NS	0.22	0.4	NS	0.07
	2	6	274	266	NS	−1.8	5.3	NS	0.03
2	1	6	8.8	20	NS	0.96	0.41	NS	0.58
	2	6	47	34	NS	0.47	0.7	NS	0.1
3	1	6	87	58	NS	−0.29	1.25	NS	0.01
	2	6	40	54	NS	0.97	1.2	NS	0.15
4	1	8	40	24	NS	0.7	0.48	NS	0.26
	2	8	45	22	NS	0.34	0.45	NS	0.09
5	1	8	27	15	NS	0.42	0.24	NS	0.34
	2	8	46	16	0.03	0.3	0.27	NS	0.17

NS = not significant ($p > 0.05$).
N = Number of 12-h observations.

Source: Wallace (1996).

collected by the stationary indoor monitor (SIM) and stationary ambient monitor (SAM) at each home. A total of ten particle samples were collected for each household (day and night samples from the PEM_{10}, SIM_{10}, $SIM_{2.5}$, SAM_{10}, and $SAM_{2.5}$). Air exchange rates were also determined for each 12-h period. Participants were asked to note activities that might involve exposures to increased particle levels. Following each of the two 12-h monitoring periods, they answered an

interviewer-administered questionnaire concerning their activities and locations during that time. A central outdoor site was maintained over the entire period (September 22, 1990 through November 9, 1990). The site had two high-volume samplers (Wedding & Assoc.) with 10-µm inlets (actual cutpoint about 9.0 µm), two dichotomous PM_{10} and $PM_{2.5}$ samplers (Sierra-Andersen) (actual cutpoint about 9.5 µm), one PEM, one PM_{10} SAM, and one $PM_{2.5}$ SAM.

Results

Of 632 permanent residences contacted, 443 (70%) completed the screening interview. Of these, 257 were asked to participate and 178 (69%) agreed.

Quality of the Data

More than 2,750 particle samples were collected, about 96% of those attempted. All filters were analyzed by X-ray fluorescence (XRF) for a suite of 40 metals. More than 1,000 12-h average air exchange rate measurements were made. A complete discussion of the quality of the data is found in Pellizzari et al. (1993) and in Thomas et al. (1993).

Concentrations

Concentrations of particles and target elements have been reported (Clayton et al., 1993; Özkaynak et al., 1993a; Pellizzari et al., 1993; Wallace et al., 1993). Population-weighted daytime personal PM_{10} concentrations averaged about 150 $\mu g/m^3$, compared to concurrent indoor and outdoor mean concentrations of about 95 $\mu g/m^3$ (Table 7-23). The overnight personal PM_{10} mean was much lower (77 $\mu g/m^3$) and more similar to the indoor (63 $\mu g/m^3$) and outdoor (86 $\mu g/m^3$) means. About 25% of the population was estimated to have exceeded the 24-h National Ambient Air Quality Standard for PM_{10} of 150 $\mu g/m^3$. Over 90% of the population exceeded the 24-h California Ambient Air Quality Standard of 50 $\mu g/m^3$.

Correlations

The central site appeared to be a moderately good estimator of outdoor particle concentrations throughout the city. Spearman correlations of the central-site concentrations

TABLE 7-23. POPULATION-WEIGHTED[a] CONCENTRATIONS AND STANDARD ERRORS ($\mu g/m^3$) PTEAM STUDY

Sample type	N	Geom. Mean	GSD	Arith. Mean ± SE	Percentile 90% ± SE	Percentile 98%
Daytime PM_{10}						
Personal	171	129	1.75	150 ± 9	260 ± 12	380
Indoor	169	78	1.88	95 ± 6	180 ± 11	240
Outdoor	165	83	1.68	94 ± 6	160 ± 7	240
Overnight PM_{10}						
Personal	168	68	1.64	77 ± 4	140 ± 10	190
Indoor	163	53	1.78	63 ± 3	120 ± 5	160
Outdoor	162	74	1.74	87 ± 4	170 ± 5	210
Daytime $PM_{2.5}$						
Indoor	173	35	2.25	48 ± 4	100 ± 7	170
Outdoor	167	38	2.07	49 ± 3	100 ± 6	170
Overnight $PM_{2.5}$						
Indoor	166	27	2.21	36 ± 2	83 ± 6	120
Outdoor	161	37	2.23	51 ± 4	120 ± 5	160

[a]Personal samples weighted to represent nonsmoking population of 139,000 Riverside residents aged 10 or above. Indoor-outdoor samples weighted to represent 61,500 homes with at least one nonsmoker aged 10 or above.

Source: Pellizzari et al. (1993).

measured by all three methods (PEM-SAM, dichot, Wedding) with outdoor near-home concentrations as measured by the SAMs ranged from 0.8 to 0.85 ($p<0.00001$). Linear regressions indicated that the central-site 12-h readings could explain 57% of the variance observed in the near-home 12-h outdoor concentrations (Figure 7-20).

Outdoor 12-h concentrations of PM_{10} could explain about 25 to 30% of the variance observed in indoor concentrations of PM_{10}, but only about 16% of the variance in 12-h personal exposures to PM_{10} (Figure 7-21). This is understandable in view of the importance of indoor activities such as smoking, cooking, dusting, and vacuuming on exposures to

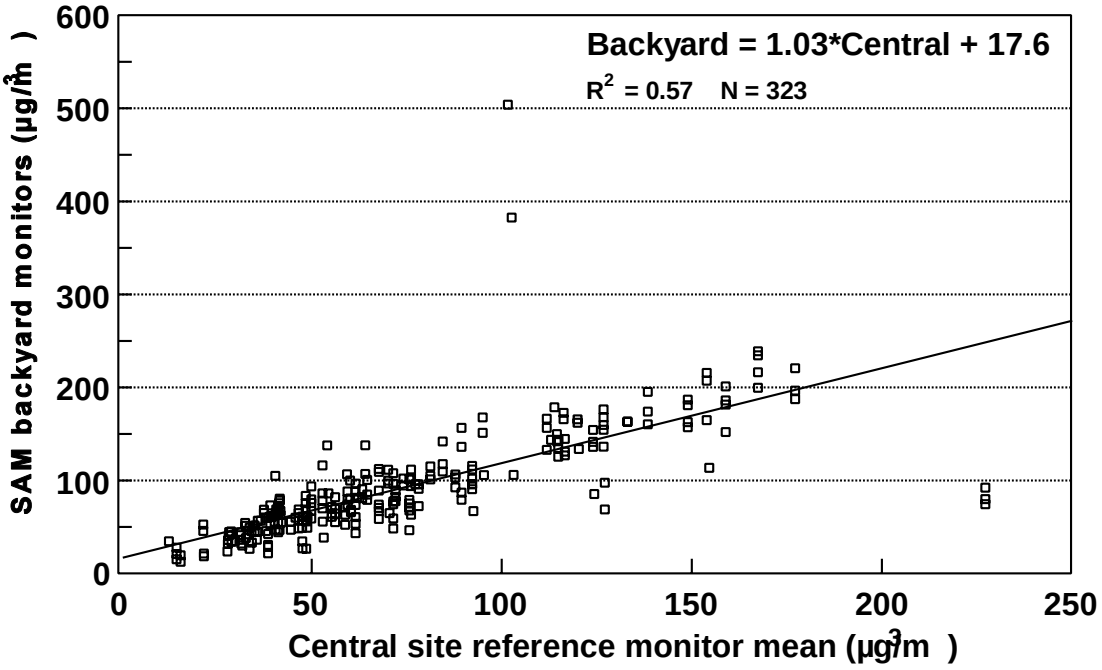

Figure 7-20. Residential outdoor monitors versus central-site mean of two dichotomous samplers in Riverside, CA. R^2 = 57%.

Source of Data: Pellizzari et al. (1993).

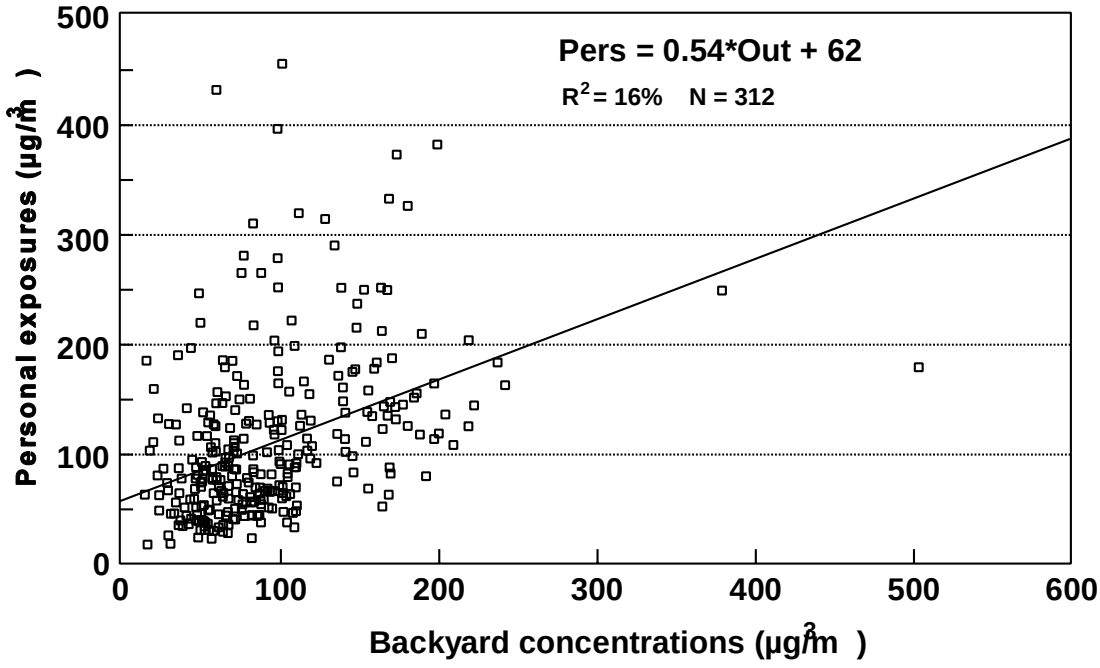

Figure 7-21. Personal exposures versus residential (back yard) outdoor PM_{10} concentrations in Riverside, CA. R^2 = 16%.

Source of Data: Pellizzari et al. (1993).

particles. The higher daytime exposures were even less well represented by the outdoor concentrations.

Indoor concentrations accounted for about half of the variance in personal exposures. However, neither the indoor concentrations alone, nor the outdoor concentrations alone, nor time-weighted averages of indoor and outdoor concentrations could do more than explain about two-thirds of the observed variance in personal exposures. The remaining portion of personal exposure is assumed to arise from personal activities or unmeasured microenvironments that are not well represented by fixed indoor or outdoor monitors.

Discussion

The more than 50% increase in daytime personal exposures compared to concurrent indoor or outdoor concentrations suggested that personal activities were important determinants of exposure. However, the nature of this "personal cloud" of particles has not yet been determined. An approach to the composition of the personal cloud is elemental analysis, using X-ray fluorescence. Analysis of all personal and indoor filters showed that 14 of 15 elements were elevated by values of 50 to 100% in the personal filters compared to the indoor filters (Figure 7-22). This observation suggests that a component of the personal cloud is an aerosol of the same general composition as the indoor aerosol. This could be particles created by activities (e.g., cooking) or re-entrained household dust from motion (walking across carpets or sitting on upholstered furniture; Thatcher and Layton, 1995). House dust is a mixture of airborne outdoor PM (primarily coarse mode), tracked-in soil and road dust, and PM produced by indoor sources. As such, it should contain crustal elements from soil, lead and bromine from automobiles, and other elements from combustion sources. This would be consistent with the observation that nearly all elements were elevated in personal samples. The lack of elevated values for sulfur may be due to the fact that submicron particles are not resuspended by human activity (Thatcher and Layton, 1995). The personal overnight samples that showed smaller mass increases than the personal daytime samples are also consistent with the fact that the participants were sleeping for much of the 12-h overnight monitoring period and were thus not engaging in these particle-generating or reentraining activities.

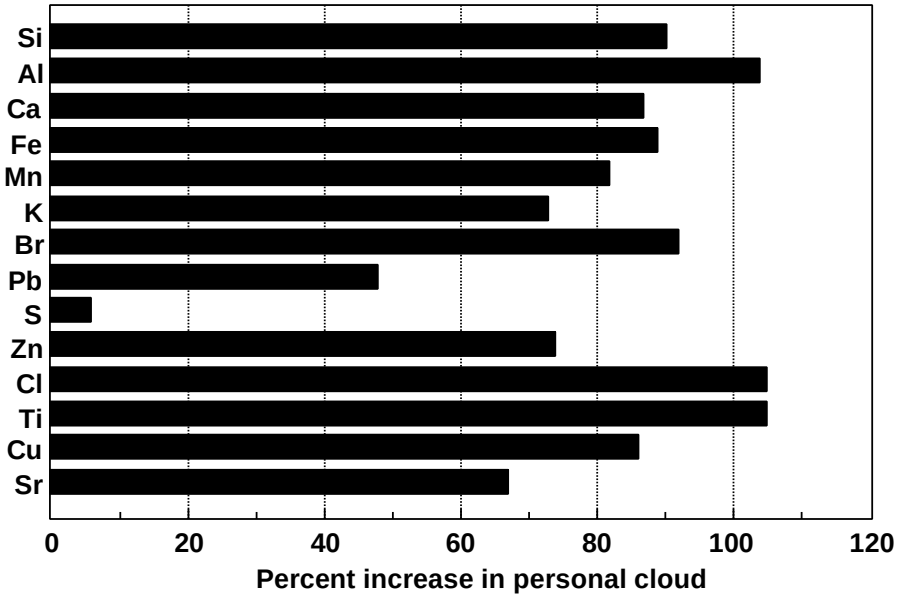

Figure 7-22. Increased concentrations of elements in the personal versus the indoor samples.

Source: Özkaynak et al. (1996).

A source apportionment of the personal PM_{10} mass during the daytime period is shown on Figure 7-23 (Özkaynak et al., 1996). This chart is derived by subtracting the average SIM and SAM (95 $\mu g/m^3$) from the mean PEM (150 $\mu g/m^3$) given on Table 7-23. The 55 $\mu g/m^3$ difference is shown as the 37% fraction of the total of 150 $\mu g/m^3$ labelled Personal 37%. The source of this "personal cloud" is indeterminable from the SIM, SAM and PEM data. As discussed previously, it is likely to consist primarily of resuspended dust that would have a composition of a mixture of all the other sources. The 15% other-indoor PM represents the indoor mass that could not be assigned to ETS, cooking or ambient PM. It is likely that the 52% of other-indoor plus personal-cloud categories contains an appreciable amount of ambient PM that came indoors over a long period of time and is resuspended by activity. If so, then the PEM would be about 50% of ambient origin.

7.4.2 Personal Exposures in International Studies

As part of World Health Organization/United Nations Environment Programme (WHO/UNEP) Global Environment Monitoring System (GEMS) activities, four pilot studies

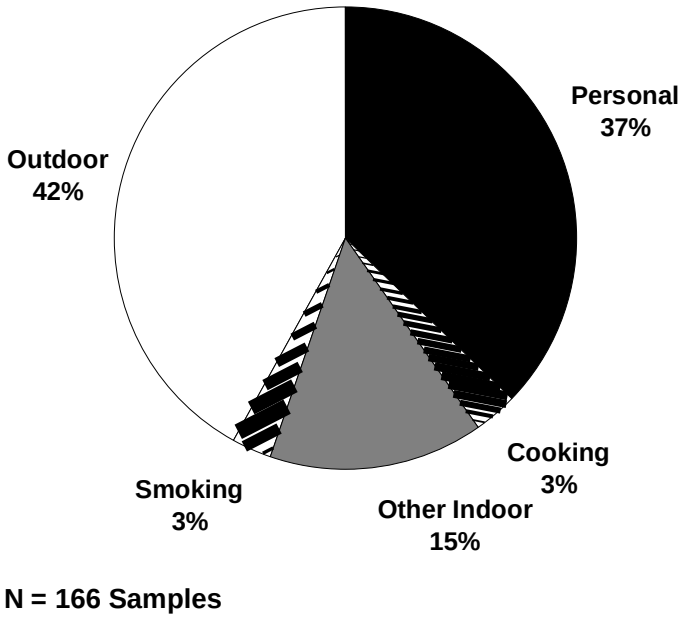

Figure 7-23. Source apportionment of PTEAM PM_{10} Personal Monitoring (PEM) Data. "Other Indoor" represents PM found by the indoor monitor (SIM), for which the source is unknown. "Personal" represents the excess PM captured by the PEM that cannot be attributed to either indoor (SIM) or outdoor (SAM).

Source: Clayton et al. (1993).

of personal exposure to PM were conducted in: Zagreb (World Health Organization, 1982a); Toronto (World Health Organization, 1982b); Bombay (World Health Organization, 1984); and Beijing (World Health Organization, 1985). In these studies, people who worked in the participating scientific institutes were recruited to carry a PM sampler, and their exposures were matched to the ambient concentrations measured outside their home or at a central station in their communities. The results of these studies, expressed as mean personal exposure (PEM) and mean ambient (SAM) concentration, and the cross-sectional regression R^2 between them are presented in Table 7-24.

The net result of these four international studies is that they appear to confirm the lack of a consistent cross-sectional relationship between individual personal PM exposures and ambient concentrations as found in the U.S. studies described in Section 7.4.1.

TABLE 7-24. SUMMARY OF WHO/UNEP GLOBAL ENVIRONMENT MONITORING SYSTEM/PERSONAL EXPOSURE PILOT STUDY RESULTS

Location Season	PM Size Cut (μm)	N	m	Time	PEM Mean ± SE	SAM Mean ± SE	R^2 PEM vs. SAM	p
Toronto	25*	13		8-h				
Winter			72		122±9	68±9	0.15	NS
Summer			78		124±4	78±4	0.10	NS
Zagreb	5	12		1-wk				
Summer			12		114±?	55±?	0.00	NS
Winter			12		187±?	193±?	0.50	NR
Bombay	3.5	15		24-h				
Winter			105		127±6	117±5	0.26	NR
Summer			102		67±3	65±3	0.20	NR
monsoon			101		58±3	51±2	0.02	NS
Beijing	3.5	20						
Winter			71	24-h	177±?	421±?	0.07	NS
Summer			40	1-wk	66±?	192±?	0.03	NS

N = number of subjects carrying personal exposure monitor (PEM).
m = total number of observations.
PEM = mean ± SD of PM concentrations (in $\mu g/m^3$) from personal exposure monitors.
SAM = mean ± SD of PM concentrations (in $\mu g/m^3$) from stationary ambient monitors.
NR = Not Reported, but listed as significant.
NS = Not significantly different from 0.
? = Not Reported.
*25 μm AD computed from flow rate and open filter design.

Source: World Health Organization (1982a,b, 1984, 1985).

7.4.2.1 Personal Exposures in Tokyo (Itabashi Ward), Japan

Tamura and Ando (1994), National Institute for Environmental Studies (1994) and Tamura et al. (1996) report results of a PM personal monitoring study conducted during 1992 in Tokyo. Seven elderly non-smoke exposed individuals who lived in traditional Japanese homes with "tatami" reed mat or carpeting on tatami or wooden flooring, and cooked with city gas, carried a PEM cascade impactor with cut-points of 2 μm and 10 μm (Sibata Science Technology, Ltd.). The seven individuals lived near the Itabashi monitoring station close to a main road. Indoor PM (SIM) and outdoor PM (SAM) were measured simultaneously for 11 48-h periods distributed in all four seasons of the year. The dataset was screened to remove observations that included indoor combustion source exposures, such as ETS from visitors, and burning of incense or mosquito coils. The reported findings were as follows:

1. The cross sectional correlation coefficient of SIM vs SAM was "relatively high" (r^2 = 0.72), but the individual coefficients for each house were higher as shown in Figure 7-24.

2. The cross sectional correlation coefficient of PEM vs SAM (measured under the eaves of the subject's house) was "relatively high" (r^2 = 0.70), but the individual coefficients for most of the subjects were higher as shown in Table 7-25.

3. The cross sectional correlation coefficient of PEM vs PM measured at the Itabashi monitoring station was slightly lower than that for the outside air (r^2 = 0.68), as shown in Figure 7-25, and the individual coefficients for most of the subjects were higher as shown in Table 7-25.

4. The individual SAM values were all linearly related with the central monitor at the Itabashi station with the coefficient of regression (R^2) in the range between 0.70 and 0.94.

5. The individual PEM values varied from 30% to 50% of the SAM values. These {PEM < SAM} data are quite different from the US data sets, such as PTEAM, where {PEM > SAM}, because they were designed to measure the influence of the outdoors on personal exposures. The difference may be due to the exclusion of ETS exposure and incense/mosquito coil burning and the Japanese customs of using reed mat (tatami) flooring and taking shoes off when entering a home. These factors would all tend to reduce the generation and resuspension of PM in the home.
   Tamura and Ando (1994) and Tamura et al. (1996) confirm the findings of Thatcher and Layton (1995) that PM < 5 μm AD has negligible resuspension in homes. Their SIM PM_2 and SIM (PM_{10} – PM_2) were highly correlated with the SAM of identical size (r = 0.879 and 0.839 respectively) but there was a negative correlation between the SIM and SAM (TSP – PM_{10}) fraction (r = – 0.036).

The importance of this study is that it demonstrates that there are very strong correlations between PEM and SAM (0.747 < r < 0.964) when the masking influences of indoor combustion sources are removed and resuspension of PM is minimized. This provides strong support to the use of an ambient monitoring station to represent the exposure of people in the community to PM of *ambient* origin.

7.4.2.2 Personal Exposures in the Netherlands

Janssen et al. (1995) preliminarily reported in an abstract results of personal PM monitoring conducted during 1994 in Amsterdam and Wageningen, NL as part of a doctoral study. Participants were 13 non-smoking adults (age 50 to 70) in Amsterdam (urban) with

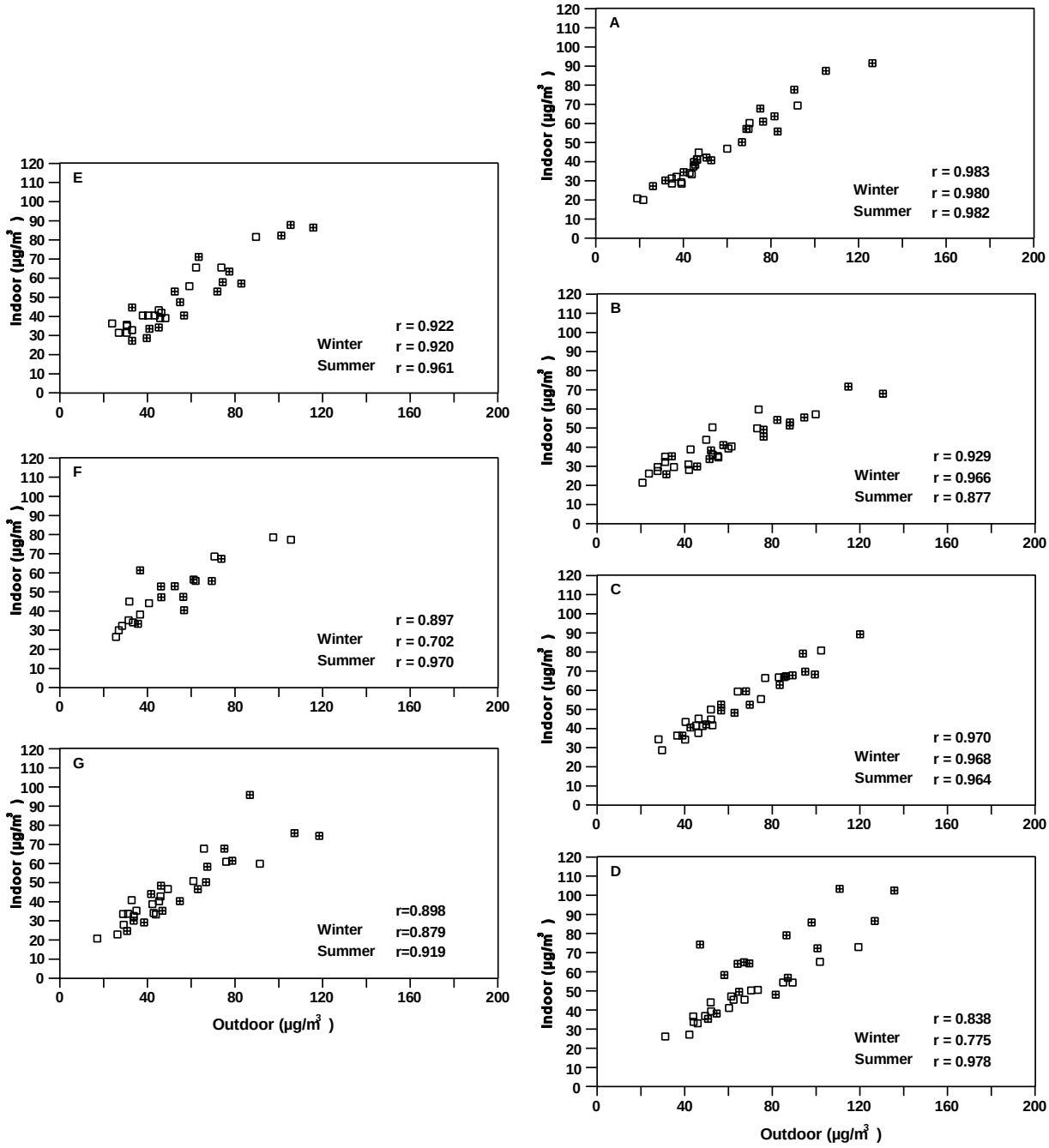

Figure 7-24. The relationship between PM_{10} in outdoor air and indoor air at each house in the study. A, B, C, D, E, F, and G, refer to the individuals identified later in Tables 7-29 and 7-30.

Source: Tamura and Ando (1994); Tamura et al., (1996).

TABLE 7-25. SUMMARY OF CORRELATIONS BETWEEN PM_{10} PERSONAL EXPOSURES OF 7 TOKYO RESIDENTS AND THE PM_{10} MEASURED OUTDOORS UNDER THE EAVES OF THEIR HOMES, AND THE PM MEASURED AT THE ITABASHI MONITORING STATION

Subject ID	Number of Samples 48-h PM_{10}	Correlation between Personal and Outdoor at home (r)	Correlation between Personal and Itabashi Station (r)
A	9	0.958	0.876
B	9	0.874	0.747
C	11	0.846	0.848
D	9	0.922	0.964
E	10	0.960	0.925
F	7	0.776	0.801
G	9	0.961	0.952
A - G	64	0.834	0.830

Source: Tamura et al. (1996).

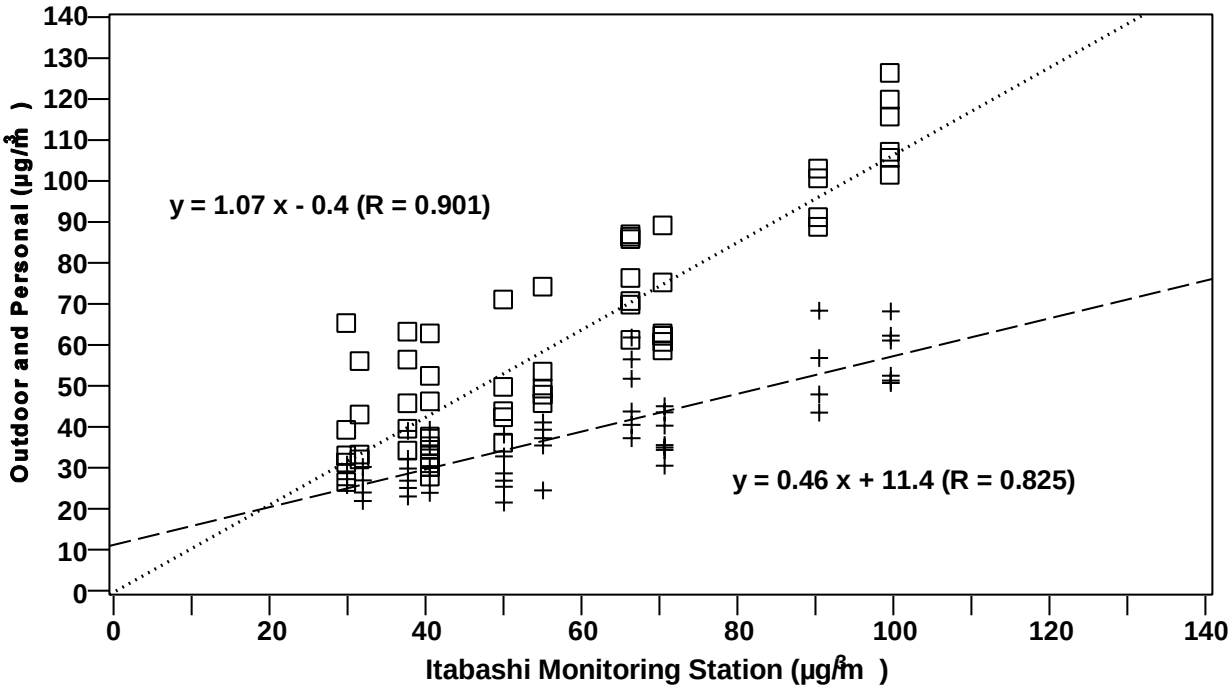

Figure 7-25. Correlations between PM_{10} at the Itabashi monitoring station and PM_{10} in outdoor and personal exposure (□=outdoor; +=personal).

Source: Tamura and Ando (1994); Tamura et al. (1996).

no occupational exposure to PM, and 15 children (age 10 to 12) in Wageningen (rural) who are presumably non-smokers. Four to eight measurements were obtained for each subject which allowed for correlating PEM and SAM within individuals (longitudinally). Only the median individual regressions were reported, as follows: adults, PEM = 26 + 0.70 SAM, R = 0.57, $R^2 = 0.32$; and children, PEM = 78 + 0.43 SAM, R = 0.67, $R^2 = 0.44$. For the children, parental smoking explained 35% of the variance between PEM and SAM. For the adults, "living near a busy road", time spent in traffic, and exposure to ETS explained 75% of the variance between PEM and SAM. The authors interpreted their preliminary results to "suggest a reasonably high correlation between personal and ambient PM_{10} within individuals". Janssen et al. (1995) also note that the low correlations observed in most of the other studies reported in the literature were cross-sectional (calculated on a group level), and were therefore mostly determined by the variation *between* subjects (e.g., ETS exposed and non-ETS exposed subjects combined in the same regression).

7.4.2.3 Reanalysis of Phillipsburg, NJ Data

With insight from the Jansen work, Wallace (1996) reanalyzed the complete Lioy et al. (1990) data from Phillipsburg, NJ, as shown partially in Table 7-20 (see also Table 7-37). Wallace (1996) compared the cross-sectional regressions of PEM on SAM for all the 14 subjects on each of the 14 days sampled, to the longitudinal regressions of each of the 14 subjects on all 14 days sampled. He found that the median R^2 (range) of the 14 individual (longitudinal) regressions was 0.46 (0.02 to 0.82); and that for the 14 daily (cross-sectional) regressions was 0.06 (0.00 to 0.39). The difference appears to indicate that, although one household may have a smoker and another not, the relationship of the indoor air in each home to the outdoor air may be the same from day to day (i.e., consistently higher than ambient in the first case, but may be consistently similar in the second). Because it provides a linkage between PEM and SAM, it bears reiteration to make certain that it is clearly understood. This PEM vs SAM relationship can be visually demonstrated with the following hypothetical example as shown on Figure 7-26a,b.

- Let two people live next door to each other at a location where the ambient PM for 5 consecutive days has a sequence {1, 2, 3, 4, 5}.

- Let person A live without ETS exposure and have a corresponding PEM series {1, 2, 3, 4, 5}, ($R^2 = 1$).

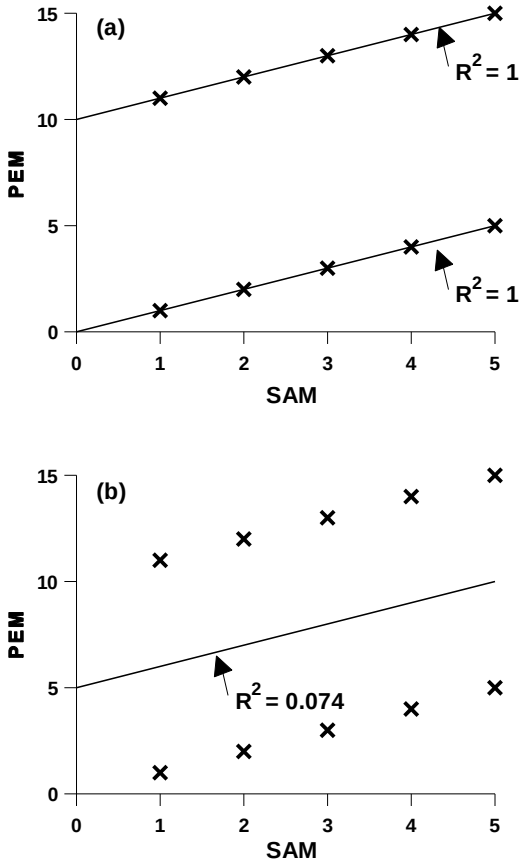

Figure 7-26. Example of difference between serial correlation (a) and cross-sectional correlation (b) of PEM and SAM, showing how pooling of individuals together can mask an underlying relationship of PEM and SAM.

- Let neighbor B live with ETS exposure and have a corresponding PEM series {11, 12, 13, 14, 15}, ($R^2 = 1$).

- When their PEM values are pooled so that they are analyzed together (cross-sectionally) {(1,11), (2,12), (3,13), (4,14), (5,15)} vs the SAM set {1, 2, 3, 4, 5}, then $R^2 = 0.074$.

- However, had the two PEM series been averaged each day, the sequence of averages {6, 7, 8, 9, 10} would have a correlation of $R^2 = 1$ with the same SAM sequence. This averaging process is described later in more detail in Section 7.6.2.

The explanation by Janssen et al. (1995) for the low cross-sectional correlations of PM PEM with PM SAM found in the literature and the new analyses reported by Tamura et al. (1996), Jansen et al. (1995), and Wallace (1996) represent a major advance in our understanding of contributions of ambient PM to personal exposures.

7.4.2.4 Overview of Comparison of Personal Exposure to Ambient PM Concentrations

The PTEAM Study and the other key PEM studies discussed in this chapter so far are summarized in Table 7-26. This table shows that many of the early studies reported no statistically significant correlation between PEM and SAM. However, these early studies were all characterized by a non-probability sample and a relatively small sample size. The PTEAM study in Riverside which was a probability sample (Clayton et al., 1993) and the Lioy et al. (1990) study in Phillipsburg, which was not a probability sample, have large sample sizes and achieved significance. The other studies, such as World Health Organization (1982a,b) or Morandi et al. (1988) are equivocal. In the following sections, PEM/SAM comparisons for some PM constituents and two means of visualizing the complex relationships of PM measured by a SAM and a PEM are developed.

7.4.3 Personal Exposures to Constituents of Particulate Matter

Suh et al. (1993) measured personal exposures to sulfate ($SO_4^{=}$) and acidity (H^+), and ambient and indoor concentrations in State College, PA, summer 1991. The correlations between personal and ambient values of sulfate and acidity were $R^2 = 0.92$ and 0.38 respectively, which is in marked contrast to the $R^2 \approx 0$ between earlier reported ambient PM and personal PM studies (Table 7-26). This relationship is supported by Figure 7-22, indicating that personal activities in the PTEAM study do not generate or resuspend sulfates less than 10 μm.

Figure 7-27 shows the consistent relation between ambient and personal sulfate measurements (slope = 0.78 ± 0.02), and Figure 7-28 shows the improvement in prediction by using the TWA with a correction factor (estimated personal sulfate = 0.885*TWA, $R^2 = 0.95$ with slope = 0.96 ± 0.02). Personal acidity was also computed by the same equation with a correction for personal ammonia (NH_3) exposure that gave an $R^2 = 0.63$. As opposed to PM which has both indoor and outdoor sources, the sulfate and acidity are virtually all of outdoor origin. Consequently, only the characteristics of the indoor environment, such as air conditioning and ammonia sources, modify the personal exposures indoors.

TABLE 7-26. COMPARISON OF PERSONAL EXPOSURE MONITOR (PEM) EXPOSURE OF INDIVIDUALS TO THE SIMULTANEOUS AMBIENT PARTICULATE MATTER (SAM) CONCENTRATION IN SEVERAL U.S. AND FOREIGN CITIES ($\mu g/m^3$)

Reference	Year	Location	PM μm	N	Time	Mean PEM	Mean SAM	R^2 PEM vs SAM	p
Binder et al.	1976	Ansonia	5	20	24-h	115	59	NS	NS
Dockery and Spengler	1981b	Watertown	3.5	18	24-h	35	17	0.00	NS
Dockery and Spengler	1981b	Steubenville	3.5	19	12-h	57	64	0.19	NR
Spengler et al.	1980	Topeka	3.5	46	12-h	30	13	0.04	NS
World Health	1982a	Toronto	25						
Organization	Winter	Non-asthmatic		13	8-h	122	68	0.15	NS
	Summer	Non-asthmatic		13	8-h	124	78	0.10	NS
	Winter	Asthmatic		13	8-h	91	54	0.00	NS
	Summer	Asthmatic		13	8-h	124	80	0.07	NS
Spengler et al.	1985	Kingston/ Harriman	3.5	97	24-h	44	18	0.00	NS
World Health	1982b	Zagreb	5	12	1-wk				
Organization	Summer					114	55	0.00	NS
	Winter					187	193	0.50	NR
Sexton et al.	1984	Waterbury	3.5	48	24-h	36	17	0.00	NS
World Health	1984	Bombay	3.5	15	24-h				
Organization	Winter					127	117	0.26	NR
	Summer					67	65	0.20	NR
	Monsoon					58	51	0.02	NS
World Health	1985	Beijing	3.5	20					
Organization	Winter				24-h	177	421	0.07	0.09
	Summer				1-wk	66	192	0.03	NS
Morandi et al.	1988	Houston	3.5	30	12-h	27	16	0.34	<0.05
Lioy et al.	1990	Phillipsburg	10	14	24-h	86	60	0.04	0.008
				14[c]	24-h	76	60	0.25	0.001
Perritt et al.	1991	Azusa	2.5	9	24-h	79	43	0.01	NS
			10	9	24-h	115	62	0.01	NS
Clayton et al.	1993	Riverside	10	141	24-h	113	84	0.23	NR
Tamura et al.	1996	Tokyo	10	7	48-h	37	56	0.68	0.000

N = Number of individuals carrying personal monitors.
NS = Not statistically significant from 0.
NR = p Value not reported, but mentioned as significant.
[a] = Year of publication.
[b] = 14 Subjects carried PEMS for 14 days for 191 valid measurements.
[c] = Three outliers are removed and regression is for 188 measurements.

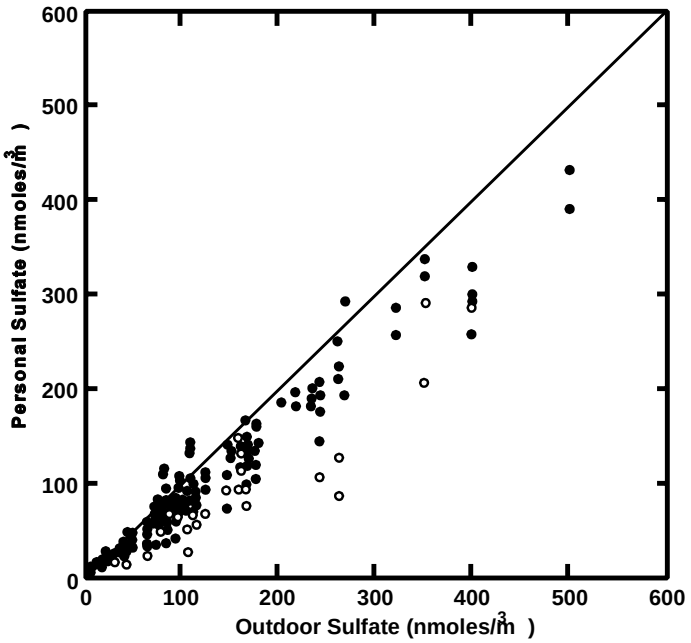

Figure 7-27. Personal versus outdoor $SO_4^=$. Open circles represent children living in air conditioned homes; the solid line is the 1:1 line.

Source: Suh et al. (1993).

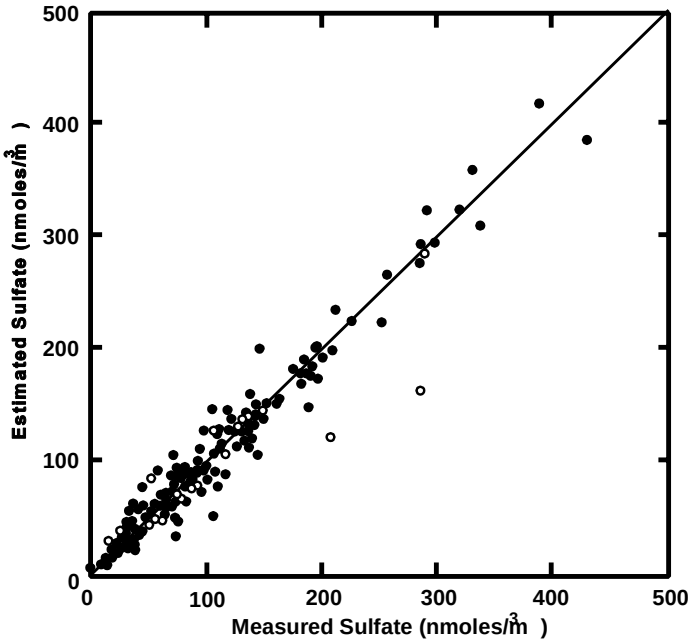

Figure 7-28. Estimated ("best fit" model) versus measured personal $SO_4^=$. Model includes indoor and outdoor concentration and activity data. Open circles are air conditioned homes; the solid line is the 1:1 line.

Source: Suh et al. (1993).

Similar high correlations for total sulfur were found by Özkaynak et al. (1996) in the PTEAM study. Regressions of personal exposures in the PM_{10} fraction on outdoor sulfur gave the following results ($\mu g/m^3$):

S_{pers} (day) = 0.62 (0.07 SE) + 0.69 (0.03) S_{out} N = 168 R^2 = 0.78

S_{pers} (night) = 0.27 (0.06) + 0.68 (0.03) S_{out} N = 162 R^2 = 0.81

Another important consideration in evaluating personal exposures, from the indoor and outdoor environmental measurements, is that the chemical composition of the excess in personal exposure compared to the TWA exposure calculation may be significantly different than that predicted from the indoor and ambient data alone.

In addition to the two factors cited just above, a microscale "personal cloud" can be generated by the person's activities which complicates the exposure measurement process. This effect is most important in occupational settings where personal exposures are not readily comparable to weighted area sampling measurements. For example, Lehmann et al. (1990) measured workers exposure to diesel engine exhaust by personal monitoring of PM_{10} with a range of 0.13 to 1.2 mg/m^3, compared to an area estimate range of 0.02 to 0.80 mg/m^3. The U.S. Centers for Disease Control (1988) reports the exposures of nurses and respiratory therapists to the aerosols of ribavirin during treatment of patients by ribavirin aerosols administered inside an oxygen tent. Bedside area monitors averaged 317 $\mu g/m^3$ while personal exposures ranged from 69 to 316 $\mu g/m^3$ with an average of 161 $\mu g/m^3$.

Environmental Tobacco Smoke (ETS) is a category of PM found in many indoor settings where smoking is taking place or recently occurred. As stated in Section 7.2, ETS is the major indoor source of PM where smoking occurs. Because of the depth of discussion of ETS in Section 7.2.2.2, no further discussion is made here other than to note that ETS adds on the order of 25 to 30 $\mu g/m^3$ to 24-h average personal exposures and residential indoor environments where smoking takes place (Holcomb, 1993; Spengler et al., 1985).

The random ETS increment will tend to reduce the correlation between PEM and SAM. If one were able to subtract out the ETS from the PEM PM data, the correlation of SAM with the non-ETS PEM PM might be improved (Dockery and Spengler, 1981b). As stated as a *caveat* in the introductory section 7.1, the inhalation of main-stream tobacco smoke will be a major additive exposure to PM for the smokers, which dwarfs the nonsmoker's PEM PM. Therefore the results presented so far apply only to nonsmokers, and a major proportion of the US

population (e.g., smokers) has a total exposure to PM that is at least one order of magnitude greater than that of the nonsmokers.

7.5 INDIRECT MEASURES OF EXPOSURE

7.5.1 Time-Weighted Averages of Exposure

The early air pollution literature related health to ambient particulate matter (TSP) concentrations as a surrogate for personal exposures to PM. Although this relationship has been shown to be highly questionable for specific individuals, it still is used in studies such as Pengelly et al. (1987) who estimated TSP exposures of school children in Hamilton, Ontario, by interpolation of ambient TSP concentrations to the school locations.

The first usage of a time-weighted-average (TWA) of environmental exposures to estimate total human personal exposure to an air pollutant (Pb) was by Fugaš et al. (1973). In theory, a human exposure to PM could be estimated by use of Equation 7-2 and knowledge of the average PM concentration while in each microenvironment (μE) that a person experiences and the duration of the exposure in each such μE (Duan, 1982; Mage, 1985). For a room with no source in operation, the whole room could be treated as a single μE. However, when a PM source is in operation and gradients exist, that very same room may need to be described by multiple μEs. These μEs could have dimensions of an order of a few centimeters close to the source and of several meters farther from the source.

Ogden et al. (1993) compared exposures from personal sampling and static area sampling data for cotton dust exposures. The British cotton dust standard specifies static sampling, because the 1960 dose-response study used to set the standard used static sampling data to compute worker exposure and dosage. Ogden et al. (1993) found median personal exposures of 2.2 mg/m^3 corresponding to a mean static background concentration of 0.5 mg/m^3. They concluded that "The presence of the body and its movement affect what a personal sampler collects, so static comparisons cannot be used to infer anything about the relationship of the (static) method with personal sampling." Ingham and Yan (1994) confirmed this finding by modelling the human body as a cylinder and showing that unless the personal monitor length/diameter ratio was greater than four, the aspiration efficiency (the fraction of particles sampled that would be sampled in the absence of the body) could be greatly affected.

Rodes et al. (1991) compared the literature relationships of personal exposure monitoring (PEM) to µE area monitoring (MEM) for PM, as shown in Figure 7-29, to which Ogden et al. (1993) is added as a single point. The authors found that PEM/MEM ratios ranged from 3 to 10 in occupational settings, and from 1.2 to 3.3 in residential settings. These combined data show that approximately 50% of all measured PEM PM values are more than 100% greater than the estimated simultaneous MEM values using the TWA approach. Their explanation points to this excess PM as due to the spatial gradient about indoor sources of PM which are usually well away from area monitors which thus fail to capture the high exposures individuals may get when in close proximity to a source. They suggest that clothing lint and skin dander could only add, at most, a few percent to the total PM mass collected by a personal exposure monitor.

The Tokyo PM_{10} data of Tamura et al. (1996), added on Figure 7-29, show that for their cohort of five elderly housewives and two male retirees that there is no evidence of a large personal cloud effect as seen in the other studies listed. Japanese people customarily take shoes off before entering a home and do not use wall-to-wall carpets, which would reduce track-in of soil and eliminate a major reservoir for resuspension of dust. However, this same cohort does display a "personal cloud" effect for the PM greater than PM_{10}, with a maximum PEM/MEM value of 3.3 for PEM = 55 µg/m^3 vs MEM 17 µg/m^3. This is consistent with the findings of Thatcher and Layton (1995) showing, on Figure 7-15, an indoor increase due to human activity, primarily for the PM greater than 10 µm in size, and Sheldon et al. (1988a,b) showing two U.S. homes for the elderly with less than 10 µg/m^3 PM_3 over a 72-h period in a nonsmoker's room.

7.5.2 Personal Exposure Models Using Time-Weighted Averages of Indoor and Outdoor Concentrations of Particulate Matter

Several studies have used the relationship of Equation 7-2 to compute the time- weighted-average (TWA) PM exposure of subjects. The procedure calls for a time-activity diary to be kept so that the time at-home, outdoors, at-work, in-traffic, etc., can be defined. By use of µE monitoring data from the study itself (or literature values of PM concentrations

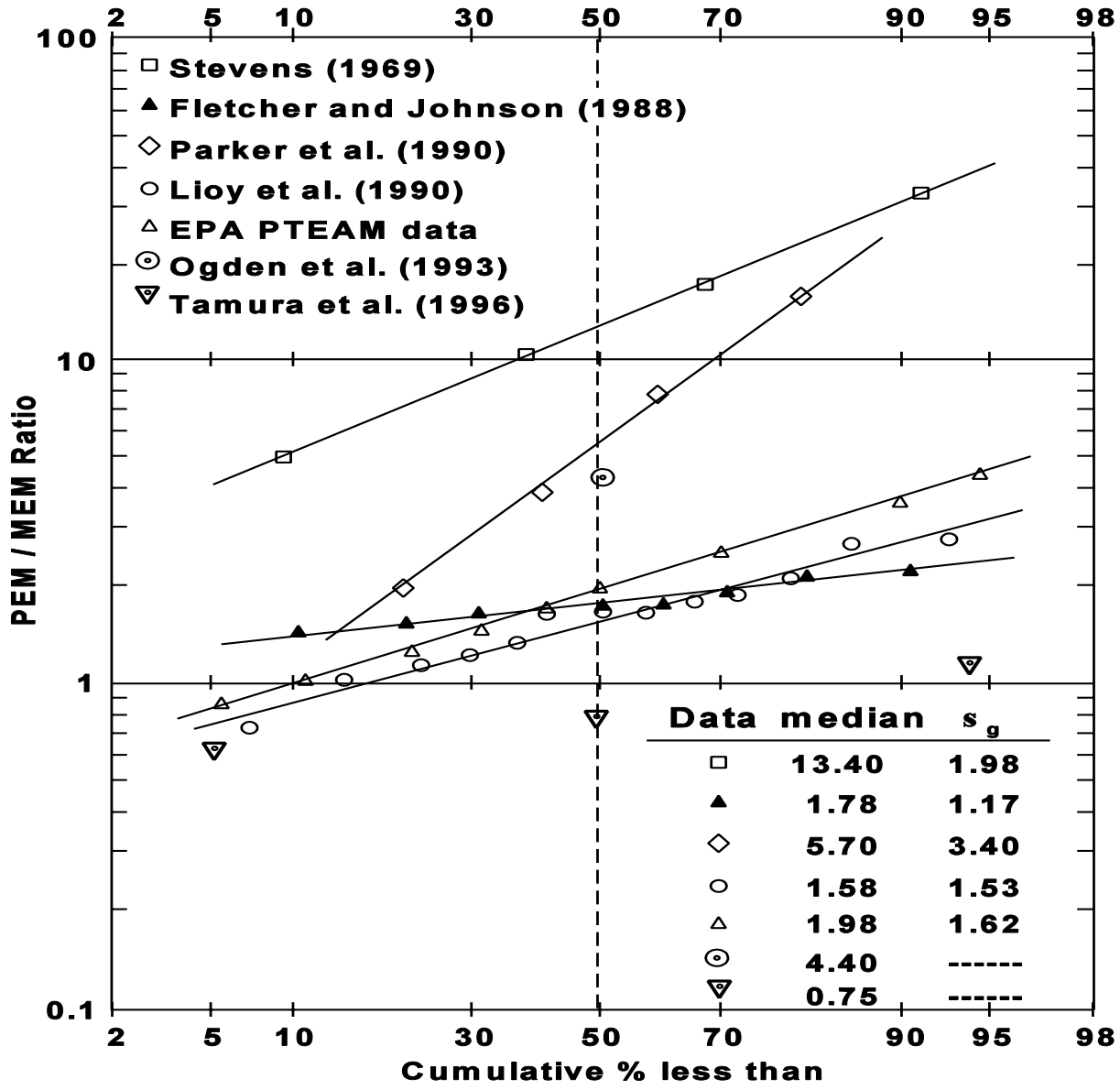

Figure 7-29. Personal activity cloud (PEM) and time-weighted average exposure (MEM).

Source: Rodes et al. (1991), Ogden et al. (1993), Tamura et al. (1996).

in similar μEs) and concurrent ambient monitoring, one can predict the concentration that would be measured if the subject had carried a PEM.

Because people in the United States spend, on average, 21 h indoors each day (U.S. Environmental Protection Agency, 1989), the concentration in indoor μEs is a most important quantity for usage within a TWA PM model. The important articles on indoor air quality for

PM have been reviewed extensively by Wallace (1996) and are covered in Section 7.2. The articles that are discussed here predict PM exposures of non-smokers that include ETS, and most provide PEM data for comparison. As opposed to the gaseous pollutants for which continuous hour-to-hour time series of SAM data are available, PM SAM monitoring data have been often only available as a time series of 24-h SAM measurements. Consequently, in much of the early PM TWA literature, the modelers assumed, by necessity, the same ambient PM in the morning and evening, which might not be accurate (Dockery and Spengler, 1981b).

Spengler et al. (1980) in a study of PEM, SAM and SIM in Topeka, Kansas, found the averages of PEM = 30 $\mu g/m^3$, SIM = 24 $\mu g/m^3$ and SAM = 13 $\mu g/m^3$. They note "It suggests that somewhere in an individual's daily activities, they are being exposed to PM at concentrations higher than what is measured either indoors or outdoors". This relationship has been found in almost all other studies, such as PTEAM (Clayton et al., 1993) where daytime PEM averaged 150 $\mu g/m^3$ and SIM and SAM averaged just under 100 $\mu g/m^3$. Spengler et al. (1985) measured 24-h PEM, SIM and SAM. The resulting relationship based on Equation 7-1 was: PEM = 17.7 $\mu g/m^3$ + 0.9 TWA. The authors noted, in addition to the previous suggestion, that the excess of PEM over TWA may be due to an incorrect assumption that the indoor and outdoor are constant during the 24-h sampling period.

Koutrakis et al. (1992), in a study discussed in Section 7.2 on Indoor Air, report that their source-apportionment mass-balance model predicts penetration from outdoors to indoors on the order of 85-90% for Pb and sulfur compounds. The authors claim that:

> "We can satisfactorily predict indoor fine aerosol mass and elemental concentrations using the respective outdoor concentrations, source type and usage, house volume and air exchange rate."

The authors further note that this may be a cost-effective approach to estimating peoples' exposure while indoors, since the necessary ambient data may be available and the housing profile may be collected with a simple interview.

Colome et al. (1992) measured indoor and outdoor PM-10 at homes of asthmatics in California. Their personal monitoring data, limited to three individuals, confirmed the relation in Figure 7-16 that "some protection from higher outdoor concentration is afforded by shelter if smokers and other particulate sources are not present". This observation may be important for estimating the exposure of elderly and infirm people who are assumed to be the susceptible cohort (Sheldon et al., 1988a,b).

Klepeis et al. (1994) present an up-to-date TWA PM Model that uses, as an input, real-time hourly PM SAM data and a mass balance equation to predict exposures of nonsmokers in various indoor settings based on ambient PM data, presence of PM sources such as smokers, and other variables relating to air exchange rates. The inclusion of the additive terms that allow for sources, such as cooking and presence of smokers adds to the TWA of Equation 7-2, which in effect is a correction for the underprediction of the μE concentration.

In summary, as described by several authors, the PM PEM exposure of individuals who are not smoke exposed has been shown to be higher than their corresponding TWA of SIM and SAM in U.S. studies. The exact reason for this excess in PM, sometimes called a "personal cloud", is not known (Rodes et al., 1991). It has been thought to reflect the fact that the person's presence itself can stir up loosely settled-dust by induced air motion and vibration (Ogden et al., 1993; Aso et al., 1993). Thatcher and Layton (1995) gave an example where merely walking into and out of a room raised the total suspended dust (PM_{10}) by 100%. A study by Litzistorf et al. (1985) of asbestos type fibers in a classroom showed how fibers (f) were stirred up when it was occupied. The levels rose from below the detectable level of 10000 f/m^3 to 80000 f/m^3 when occupied, and they returned to below detectable levels within 1 h after the end of the class. Millette and Hays (1994) present a detailed discussion of the general topic of resuspended dust in their text on settled asbestos dust.

It may not be a proper procedure to use a 24-h average concentration in a physical setting, such as a kitchen, to estimate a person's exposure while in the kitchen. As described previously in the discussion of the definition of a microenvironment in Section 7.1.2, the same kitchen can constitute one or more μEs depending on the source operation pattern. In many studies, such as Spengler et al. (1985), the SIM sampled the indoor residential setting for 24-h in phase with the PEM. The resulting average SIM will often underestimate the person's exposure while they are at home and may contribute to the difference between a TWA exposure and the PEM.

In a similar manner, a person's workplace exposure may be more or less than that in their home. In the PTEAM study (Clayton et al., 1993), there was a general decrease in exposure for those employed outside their home. However, employment in a "dusty trade", such as welding, may increase their PM PEM. Lioy et al. (1990) give an example of a subject with a hobby involving welding having a 24-h PEM reading of 971 $\mu g/m^3$.

Indirect estimation of a person's time-weighted-average (TWA) PM exposure may be a cost-effective alternative to direct PEM PM measurement. Mage (1991) compared the advantages and disadvantages of the TWA indirect method compared to the direct PEM method. The primary advantages of the indirect method are the lower cost and lower burden on the subject, because it uses only a time-activity diary and no PM PEM is required; the disadvantage is the lower accuracy. The primary advantage of the PEM PM method is that it is a higher accuracy direct measurement; the main disadvantages are the higher cost and higher burden on the subject (see Section 7.3.1). Mage (1991) proposed a combined study design in which direct measurements on a subset of subjects can be used to calibrate the TWA estimates of other subjects. Duan and Mage (1996) present an expression for the optimum fraction of subjects to carry the PEM as a function of the relative cost of the PM PEM to the TWA PM estimate and the correlation coefficient between the PM PEM data and the PM TWA estimates.

7.6 DISCUSSION

7.6.1 Relation of Individual Exposures to Ambient Concentration

The previous sections discussed the individual PM PEM vs PM SAM relationships of the studies listed in Table 7-26. In many of the cross-sectional PM studies, no statistically significant linear relationship was found between PEM and SAM, but in some other studies the relationship is positive and statistically significant. However, as shown by Lioy et al. (1990), Janssen et al. (1995), and Tamura et al. (1996), the serial correlations between PEM and SAM within an individual's time series are often highly positive and significant. This section discusses these data in terms of understanding the complex relationship between the SAM concentrations and the *individual* PEM exposures. In the following section, the relationship of the SAM to the *mean* PEM in the community surrounding the SAM will be presented.

The principle of superposition is offered as a basis for visualization of the process involved in creating a total exposure. A linear system will exist for respirable-PM PEM exposures if the expected PEM response to a source emitting 2 mg/min of PM is exactly twice the PEM response to that identical source emitting 1 mg/min of identical PM. If superposition applies, then we can construct the total exposure by adding all the increments of exposures from the various source classes and activities that a subject performs on a given day.

Let the SAM be representative of the macroscale ambient PM concentration in the community as shown on Figure 7-30a. This is the exposure that would be measured for a person if they spent 24-h per day outdoors near the SAM site. Neglecting local microscale variation (e.g. backyard barbecue or leaf burning), while people are outdoors they are exposed to 100% of the SAM value (Figure 7-30b). Assume that this exposure is also the baseline PM for a location in traffic which occurs outdoors. The increment produced by the local traffic is considered later.

While people are indoors, they are exposed to a variable fraction of time-lagged SAM PM. This constitutes an amount of (1) the fresh PM which depends on recent SAM and the air exchange rate between indoors and outdoors, and the PM deposition sinks (filtration of recirculated air, surfaces, etc.), and (2) PM from outdoor sources that had been deposited in the past but is resuspended due to human activity and air currents. PTEAM (Özkaynak et al., 1996), as cited in Section 7.2, found that outdoor air was the major source of indoor particles, accounting for 75% of the fine fraction (<2.5 μm AD) and 67% of the thoracic fraction (< 10 μm AD) in indoor air. It is noted that these average fractions will be lower in communities with lower average SAM values. Lewis (1991) reported an apportionment of indoor air PM in 10 homes within a wood burning community in Boise, ID. The results showed that 50% of the fine PM was of outdoor origin (SAM), and in 9 of 10 homes, 90% of the sulfur was from outdoors (one home had an anomalous sulfate injection from a humidifier using tap water). This is consistent with indoor sources varying independently of the SAM in a stationary manner (constant mean and variance), so that the relative contribution of indoor sources to indoor exposures decreases as SAM increases. Figure 7-30c represents the increment to PEM from outdoor sources of SAM while the

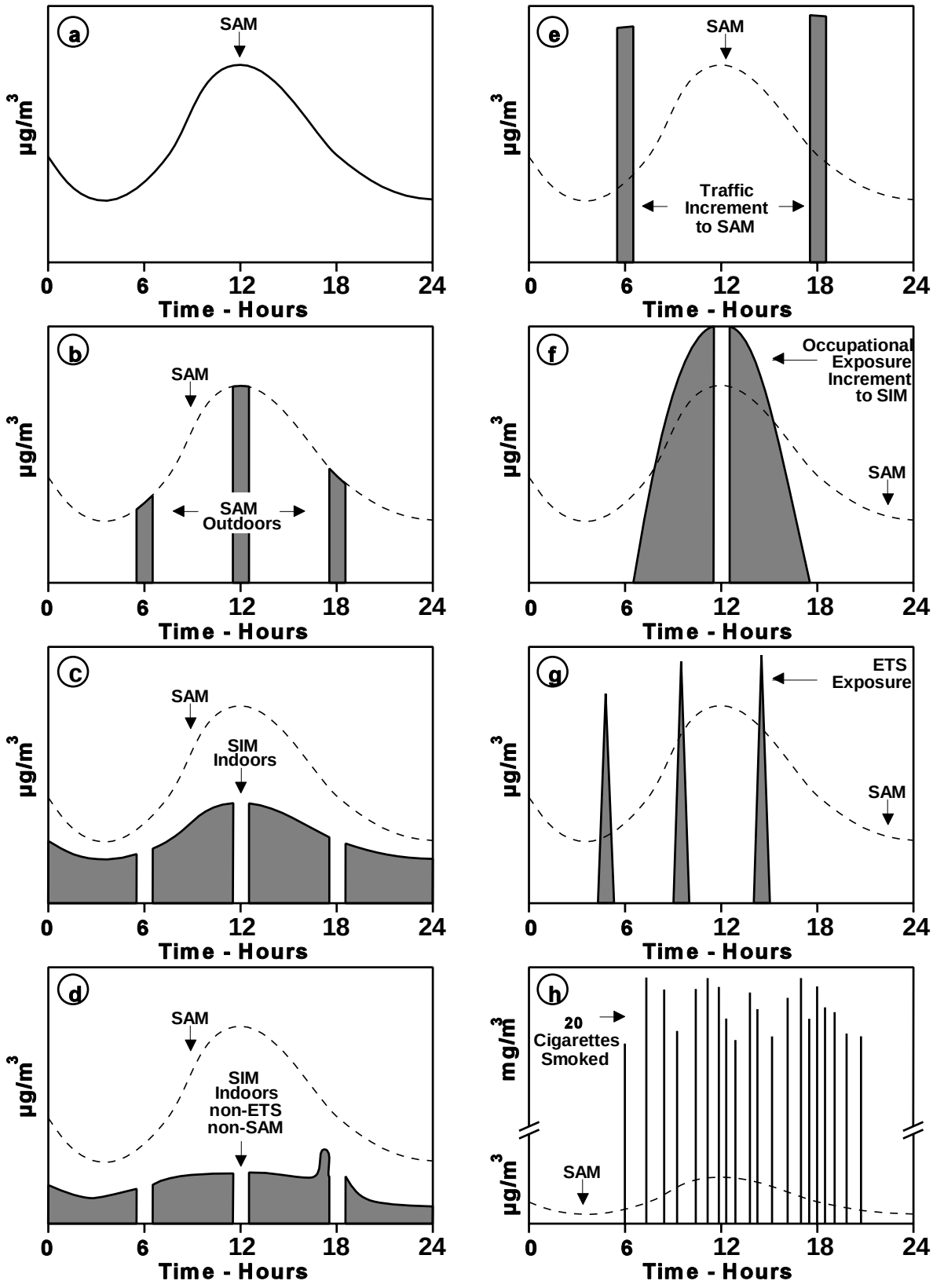

Figure 7-30. Components of personal exposure.

subjects are indoors at home and at work. The SAM value is shown as the dotted line for reference in this and all the following Figures 7-30c to 7-30h.

While people are indoors, at home, and at work, they also are exposed to PM emitted by indoor sources - *other than* ETS from passive smoking and specific occupational sources. These sources, such as cooking, lint from clothing and furnishings, mold, insects, etc., create PM that agglomerates and deposits as visible dust that can be continuously resuspended, which constitutes an additional PEM increment. Figure 7-30d shows the additive effect of this source. In traffic, or near vehicles in a parking garage or parking lot, people are exposed to an increment of PM over and above the SAM value for that location. Figure 7-30e shows the additive PM for this setting that would be added to Figure 7-30b for the local vehicular emissions.

At work in a "dusty trade" (e.g., welder, mechanic, or miner) there is an increment of exposure associated with these occupational activities that generate PM. Figure 7-30f represents the additive PM for these activities which are assumed to take place "indoors".

In an indoor setting, in the presence of a smoker or the wake of a smoker, a PEM will record an increment of ETS associated with the act of smoking. Figure 7-30g shows the added PM increment for this source.

Last, but not least, is the physical act of smoking itself. As described previously, the main stream smoke from a cigarette, cigar, or pipe is inhaled directly without being sampled by a PEM. The mass of PM directly inhaled from smoking one-pack-per-day of cigarettes rated as delivering "1 mg `tar' per cigarette by FTC method" is 20 mg per day (Federal Trade Commission, 1994). If this were distributed into a nominal 20 m^3 of air inhaled per day, it would be an additive increment on the order of 1 mg/m^3 to a 24-h PEM reading. Tar emissions as rated by the Federal Trade Commission (1994) range from <0.5 mg/cigarette to 27 mg/cigarette. Therefore one-pack-per-day smokers can have a PM exposure standard deviation that is much larger than the mean exposure to PM of non-smokers, simply from choice of brand. Figure 7-30h represents the impact of the act of smoking as creating exposures represented by the vertical spikes with an integral area $\geq$ 1 $mg\text{-}day/m^3$ per day.

For all subjects, by the principle of superposition, the sum of the areas shown in Figures 7-30b and 7-30c represents the exposure of an individual to the PM constituents that are characterized by a SAM PM concentration. The additional exposure categories that are independent of the SAM concentration (Figures 7-30d through 7-30g) and are appropriate for

that subject would represent the portion of 24-h PEM PM that is not associated with SAM. Variance of SAM should explain much of the variance in the SAM related PEM fraction as defined by Figures 7-30b and 7-30c. The summation over a full day for all categories 7-30b to 7-30g would be the PEM for any subject, such as is shown in Figure 7-2 (Repace and Lowery, 1980).

Although there are no data for PEM PM exposures of individuals living in homes without any indoor sources of PM, there are data for PEM sulfate as discussed previously in Section 7.4.3. Given that there are negligible sources of sulfur (S) that originate in the home (matches, low-grade kerosene, humidifiers using tap water), the high correlation of PEM sulfate and SAM sulfate ($R^2 = 0.92$) of Figure 7-27 reported by Suh et al. (1993), where no appreciable sources of S were present, is an indication that the same relationship should hold for all SAM PM of that size range. The data of Anuszewski et al. (1992) show that light scattering particles measured by nephelometry had a very high correlation between indoor and outdoor concentrations ($R^2 > 0.9$) for one home, but were lower for others. Lewis (1991) and Cupitt et al. (1994) report that PM_{10} appears to penetrate with an average factor of 0.5 in Boise homes without woodburning. The factor goes up to 0.7 with woodburning, and the authors assume that the factor would go up to 0.9 in the summer when homes are less tightly sealed. However, the authors did not consider the deposition rate *k*. This is in contrast to the data of Thatcher and Layton (1995), who measured *k* and found penetration factors of 1.0 for all PM sizes < 10 μm.

If the variance of the PEM PM portion which is uncorrelated to SAM (Figure 7-30d to 7-30g) is very large, the percentage of the variance of the PEM PM that can be explained by the variance of SAM PM will be very small. It may be possible that the different populations sampled, cited in the studies of Table 7-26, have widely different home characteristics, occupations, mode of commuting, and smoking exposures that contribute to the different PEM vs SAM relationships. In some of the cleaner communities (such as Watertown, MA; Topeka, KS; Waterbury, VT; and Kingston and Harriman, TN) SAM averaged less than 20 $\mu g/m^3$. The non-SAM increments to PEM exposure in these locales were greater than the SAM and may have been so variable between people (eg. ETS and non-ETS exposures pooled together) that the PEM PM became insignificantly correlated with the SAM PM data. The exception is Houston, TX, with a SAM = 16 $\mu g/m^3$ and a significant $R^2 = 0.34$ ($0.005 < p < 0.05$). However, Morandi et al. (1988) note that deletion of two outlier observations would reduce R^2 and make it

nonsignificantly different from 0 ($p > 0.2$). This is in contrast to the three studies in communities with high SAM levels (Tamura et al., 1996; Clayton et al., 1993; Lioy et al., 1990), where the relations between PEM and SAM were significant.

All discussions above relate to nonsmokers. As for the smoker, the exposure from Figure 7-30h would outweigh the sum of all the other exposures, 7-30b through 7-30g. This smoking increment may have an important implication for interpretation of epidemiology studies that relate ambient PM, as a surrogate of exposure, to mortality or morbidity.

Because the daily amount of individual smoking and other exposures from indoor sources (cooking, ETS, resuspension of settled dust by walking into carpeted rooms, hobbies) is independent of the daily SAM value, the variance of the PM SAM value is a surrogate for the variance component of total personal exposures to PM associated with PM SAM. For nonsmokers ambient PM reflects about 50 to 70% of their PM_{10} exposure that by definition does not contain directly inhaled smoke exposure (Tamura et al., 1996; Özkaynak et al., 1996). This relationship would also hold for the total PM exposure of smokers minus the effective increment they receive from their direct smoking which is independent of PM SAM. Therefore, a relationship between ambient PM (SAM) and human exposure to PM (PEM) that makes sense, is that the SAM value is a surrogate for personal exposure to PM (PEM) from PM originating in the ambient air. This relationship would apply to everyone, smokers and nonsmokers alike. However, treating SAM as a surrogate for total personal exposure to PM from all sources, including those major sources of PM that vary independently of SAM (active smoking and occupational exposures), would be wrong.

7.6.2 Relation of Community Particulate Matter Exposure to Ambient Particulate Matter Concentration

For the morbidity/mortality studies described in Chapter 12 that use SAM as the independent variable, that SAM can be interpreted to stand as a surrogate for the average community exposure to PM from sources that influence the SAM data. These sources of ambient PM do not include indoor sources such as the "personal cloud" of skin flakes and lint, ETS, cooking fumes, and resuspended PM from walking on a dirty carpet. Thus, if we could subtract off from each PEM measurement the contribution to the total exposure from the indoor sources, such as smoking, cooking, carpets, and personal clouds, the residual PM from ambient

sources would probably improve the correlation with SAM, as described by the data of Tamura et al. (1996) for nonsmoking-noncarpeted homes occupied by elderly people. Mage and Buckley (1995) tested the relationship of the mean PEM to SAM as a means to minimize the affect of variations of these indoor sources of PM on the relation of PEM to SAM, and their results, with modifications, are presented in the following section.

There are several different models for these analyses and although most describe the same linear relationship, the models differ greatly in their assumptions about the error terms. The discussion of the various models is followed by U.S. EPA reanalyses of five different PEM-SAM data sets described previously in Section 7.4.

7.6.2.1 Methodology

Methods for Missing Data

One common difficulty in the use of aerometric data is the presence of missing data elements. For example, consider the following PEM data from the study of Tamura et al. (1996). The authors measured the 48-h personal exposure to PM_{10} for seven individuals living near a main road for 11 periods in four seasons distributed over a complete year. This example has a great deal of missing data, and for purposes of computation, the data were split into a group living close to the road (persons A, B, C, and D), and a group living farther from the road (persons E, F, and G). Their indoor and outdoor data were shown previously on Figure 7-24. The PEM data for the first group are shown in Table 7-27.

Unless pairwise correlations are computed, the standard solution to the problem is to delete all observations for which any of the variables are missing. This approach, known as a complete-case analysis, is standard in the majority of the statistical packages. For this example, we would be left with only 5 of the original 11 periods of observation. This section will describe a model which will allow for the inclusion of all available data.

The reason for the missingness of the data is extremely important because it determines our ability to obtain maximum likelihood estimates (MLE). The following definitions are paraphrased from Little and Rubin (1987): If the probability of being missing is independent

TABLE 7-27. 48-HOUR PERSONAL EXPOSURE TO PM_{10} ($\mu g/m^3$)
(Data Taken by Subjects Living Along a Main Road in Tokyo)

Period	Person A	Person B	Person C	Person D
1	43.7	40.4	37.5	52.3
2	27.4	31.5	29.8	26.0
3	30.2	39.2	32.7	M
4	22.4	29.2	25.9	38.2
5	57.4	43.2	43.3	M
6	M	26.1	27.9	39.9
7	M	37.9	35.8	34.6
8	24.6	M	41.4	39.8
9	31.0	34.5	36.0	45.6
10	22.9	M	24.3	30.6
11	68.7	51.8	52.6	68.1

M = Missing observation.

Source: Tamura et al. (1996).

of both the variables missing and the variables present, then the data are said to be missing completely at random (MCAR). If the probability of being missing depends on the variables present, but not on the variables missing, then the data are said to be missing at random (MAR). If neither situation holds, then there are no general solutions to the problem. This would happen if the value of the missing variable (which is not known to us) is directly related to its probability of being missing. Laird (1988) discusses models used for maximum likelihood estimation with missing data, as well as a detailed discussion of the non-response mechanism.

One solution is to assume that the measurements are distributed as a multivariate normal distribution (or to assume that some transformation of the data give a multivariate normal distribution). The estimation of the parameters of a multivariate normal model with missing data is a problem which has been discussed for many years (see Afifi and Elashoff, 1966). The first general solution to the problem of estimating a mean vector and covariance matrix from a multivariate normal distribution with data missing at random was given by Woodbury and Hasselblad (1970). The solution, referred to as the "Missing Information Principle", was generalized to other missing data problems by Orchard and Woodbury (1972). Proof that the

method always improved the likelihood was given by Dempster et al. (1977), and the generalized solution method was named the E-M algorithm.

To describe the problem, the following notation will be used. Let $\mathbf{x} = x_1, x_2, \ldots, x_k$ be a k-dimensional random vector from a multivariate normal distribution

$$f(x|\mu,\Sigma = (2\pi)^{-k/2}|\Sigma|^{-1/2}e^{-(x-\mu)\Sigma^{-1}(x-\mu)/2} \tag{7-10}$$

where $\sum$ is a symmetric positive definite matrix and $\boldsymbol{\mu}$ is a vector. The mean of the vector $\mathbf{x}$ is $\boldsymbol{\mu}$ and its covariance is $\sum$. Assume that we have n observations from this distribution, $\mathbf{X}_1, \mathbf{X}_2, \ldots, \mathbf{X}_n$.

The E-M algorithm can be used to estimate the parameters of a multivariate normal distribution. The method starts with any reasonable first estimate of the parameters. Assume that we have initial estimates of the parameters $\boldsymbol{\mu}$ and $\sum$, which can be obtained by filling in the missing data with the column means and then estimating the parameters in the usual manner. The E step consists of estimating the sufficient statistics. For this model, the sufficient statistics are the sums and sums of squares of cross products.

Assume that at one particular point, $\mathbf{X}_i$, some of the observations are missing and some of the observations are present. Without loss of generality, we will drop the subscript, i, and rearrange the subscripts so that the vector $\mathbf{X}$ is $[\mathbf{X}_1, \mathbf{X}_2]$ where all of the observations, $\mathbf{X}_1$, are missing and all the observations $\mathbf{X}_2$ are present. Partition the mean vector $\boldsymbol{\mu}$ and the covariance matrix $\boldsymbol{\Sigma}$ in a similar fashion

$$\mu = \begin{bmatrix} \mu_1 \\ \mu_2 \end{bmatrix} \text{ and } \Sigma = \begin{bmatrix} \Sigma_{11} & \Sigma_{12} \\ \Sigma_{21} & \Sigma_{22} \end{bmatrix}. \tag{7-11}$$

Compute the regression of the missing observations on the observations present

$$\beta = \Sigma_{12}\Sigma_{22}^{-1}. \tag{7-12}$$

Estimate the missing values, $\mathbf{X}_1$, by their expected values

$$E(X_1) = \mu_1 + \beta(X_2 - \mu_2). \tag{7-13}$$

Compute the correction to the expected sums of squares

$$\Sigma_{11|2} = \Sigma_{11} - \Sigma_{12}\Sigma_{22}^{-1}\Sigma_{21} . \quad (7\text{-}14)$$

Now add the vector **X** to the sums and **XX'** to the sums of squares and cross products using their expected values for the missing values; remember to add $\Sigma_{11|2}$ to the cross products corresponding to $\mathbf{X}_1$.

The M step consists of recomputing the estimates of $\boldsymbol{\mu}$ and $\sum$ from the completed sums and sums of squares and cross products. This procedure will converge, typically taking five to 20 iterations for a moderately sized problem. Using the methods just described, the estimates of both the missing values and the parameters for the data of Tamura et al. (1996), based on U.S. EPA reanalyses, are shown in Table 7-28.

This method was also used to fill in the missing values for persons E, F, and G (shown in Table 7-29). Once the missing data were estimated, the average across all seven persons was computed and compared with the ambient measurement monitor as shown in Table 7-30. These data will be used as examples for the next section.

Linear Regression Models

The various linear regression models are illustrated next using the average personal exposure values from the Tamura et al. (1996) data set which were described in the previous section. For these examples, the average personal exposure will be considered the dependent variable and the ambient concentration at the Itabashi site will be the independent variable.

The first model is often referred to as the fixed independent variable model (see Dunn and Clark, 1974, p. 225). The model assumes that the dependent variable is a linear function of the independent variable with random error which is normally distributed (this is a bad assumption but this is the most commonly used model). This can be written as

TABLE 7-28. PARAMETER ESTIMATES FOR 48-HOUR PM_{10} PERSONAL EXPOSURE MONITOR DATA TAKEN BY SUBJECTS LIVING NEAR A MAIN ROAD IN TOKYO ($\mu g/m^3$)
(Estimated Missing Values Shown in Parentheses)

Day	Person A	Person B	Person C	Person D
1	43.7	40.4	37.5	52.3
2	27.4	31.5	29.8	26.0
3	30.2	39.2	32.7	(37.4)
4	22.4	29.2	25.9	38.2
5	57.4	43.2	43.3	(58.4)
6	(29.3)	26.1	27.9	39.9
7	(28.9)	37.9	35.8	34.6
8	24.6	(43.3)	41.4	39.8
9	31.0	34.5	36.0	45.6
10	22.9	(26.7)	24.3	30.6
11	68.7	51.8	52.6	68.1
Means	35.1	36.7	35.2	42.8
	Covariance/Correlation Matrix (Correlation below diagonal)			
Person A	215.8	83.9	96.4	157.4
Person B	0.745	58.9	58.4	67.6
Person C	0.819	0.949	64.3	79.0
Person D	0.888	0.731	0.816	145.6

Source: Parameter estimates, including the calculation of estimated missing values, and covariance/correlation matrix results from reanalyses by U.S. EPA of data from Tamura et al. (1996).

$$Y_i = \beta_0 + \beta_1 X_i + \varepsilon_i, \text{ where} \quad (7\text{-}15)$$

$i = 1,2,\ldots,n$, n is the number of observations, and ϵ_i is normal with mean 0 and variance σ^2. No assumption is made about the distribution of the independent variable since it is considered to be fixed.

Using the previous example, the estimated coefficients are given in Table 7-31, and the results are shown graphically in Figure 7-31.

The second model is often referred to as the bivariate normal model (see Dunn and Clark, 1974, p. 239). This model assumes that the dependent variable and the independent variable are both normally distributed. Actually, the assumption is stronger—it assumes that

TABLE 7-29. PARAMETER ESTIMATES FOR 48-H PM_{10} PERSONAL EXPOSURE MONITOR DATA TAKEN BY SUBJECTS LIVING FARTHER FROM THE SAME TOKYO MAIN ROAD DESCRIBED IN TABLE 7-28 (in $\mu g/m^3$) (Estimated Missing Values Shown in Parentheses)

Period	Person E	Person F	Person G
1	57.1	62.2	(37.1)
2	(30.9)	26.5	(29.0)
3	26.8	23.1	25.3
4	32.9	(30.6)	27.2
5	68.6	(69.2)	48.0
6	31.2	26.6	24.4
7	26.5	24.0	29.7
8	35.8	(28.7)	37.7
9	40.7	(36.9)	35.4
10	29.8	27.5	22.4
11	62.5	51.2	61.0

Source: Parameter estimates, including the calculation of estimated missing values, based on reanalyses by U.S. EPA of data from Tamura et al. (1996).

the joint distribution of the two variables is bivariate normal. The bivariate normal distribution is a special case of the multivariate normal distribution described earlier. The intercept, β_0, and regression coefficient, β_1, are estimated by the same formulas as were used in the first model even though the assumption is not the same. The R-squared term is also the same, but the ANOVA Table no longer makes any sense.

The third linear model is the same as the first except that a lognormal error term is used. This kind of model requires the use of a general linear model fitting routine. The model gives less weight to large deviations about the predicted line where the predicted values are already large. The model still assumes that the independent variable is fixed and measured without error. The fit to the previous example is shown in Table 7-32. There is no measure comparable to R^2, but the log-likelihoods can be compared directly. Note that

TABLE 7-30. AVERAGE PERSONAL EXPOSURE DATA COMPARED WITH ITABASHI SITE MONITOR (PM_{10}; $\mu g/m^3$)

Period	Itabashi Site	Average Personal
1	66.5	47.2
2	30.1	28.7
3	37.9	30.7
4	50.3	29.5
5	90.5	55.4
6	40.7	29.3
7	40.5	31.1
8	55.1	35.9
9	70.6	37.2
10	31.9	26.3
11	99.5	59.4

Source: Data from Tamura et al. (1996).

TABLE 7-31. RESULTS OF LINEAR REGRESSION ANALYSIS, ASSUMING A NORMAL ERROR USING THE EXPOSURE DATA FROM JAPAN

Linear regression
Y = intercept + slope X

Variable	Beta	Std. Err. Beta
Intercept	11.32	3.025
Slope	0.466	0.050

ANOVA Table

Source	Sum of Squares	Mean Square Error	D.F.	F-value
Regression	1194.3	597.2	2	42.9
Error	125.3	13.9	9	
TOTAL	1319.6	120.0	11	

R-squared = 0.905
Log-likelihood = −28.99

Source: U.S. EPA reanalyses of data from Tamura et al. (1996).

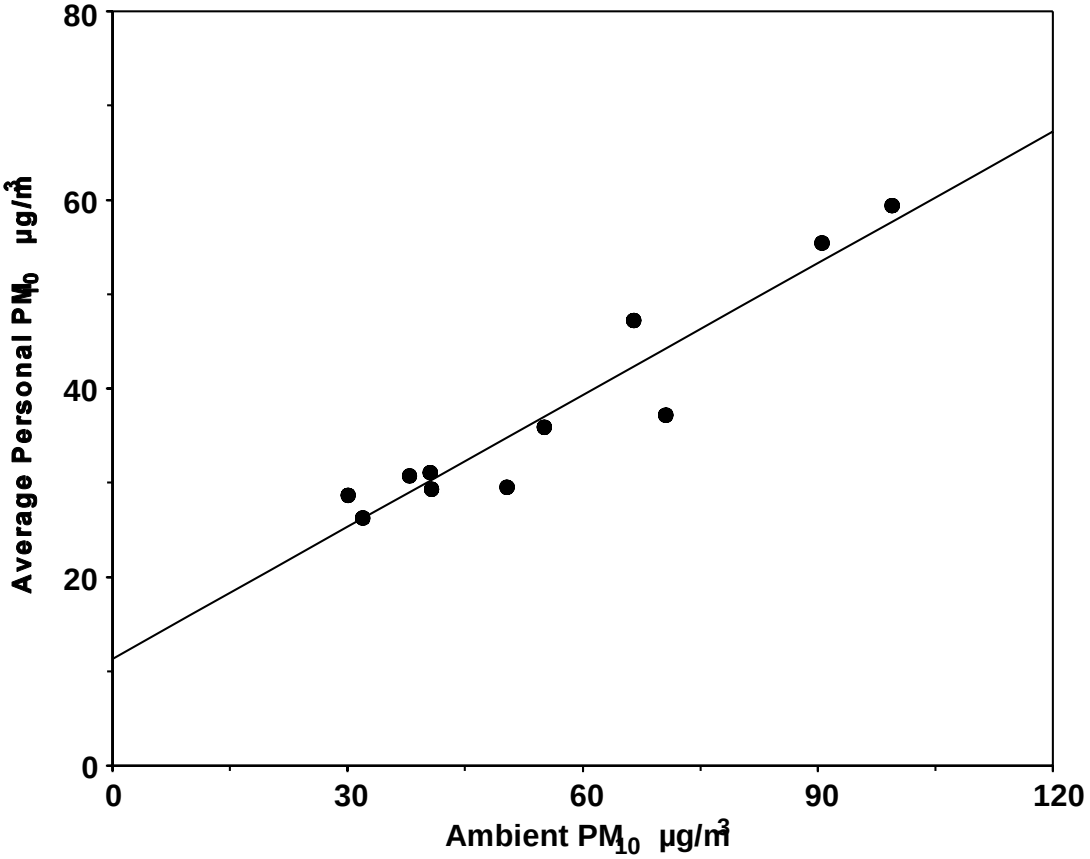

Figure 7-31. Plot of 48-h average personal PM_{10} exposure and ambient PM_{10} data from Japan—linear regression.

Source: U.S. EPA reanalyses of data from Tamura et al. (1996).

TABLE 7-32. RESULTS OF LINEAR REGRESSION ANALYSIS, ASSUMING A LOGNORMAL ERROR USING THE EXPOSURE DATA FROM JAPAN

Multiple log-linear regression analysis

Variable	Mean	Beta	Std.Err.Beta
Ambient	55.78	0.43	0.06
Mean	1	13.07	3.26

Sum of squares for error = 0.089

Mean square error = 0.010, d.f. = 9

Log-likelihood = −28.50

Source: U.S. EPA reanalyses of data from Tamura et al. (1996).

the linear model with a lognormal error fits slightly better than the normal error model, although the difference of 0.49 in the log-likelihood is not statistically significant.

Orthogonal Regression Models

Orthogonal regression is also known as principle components regression. There is no real assumption about the model. The purpose of the analysis is to pass a line through the data such that as much of the variation is explained as possible. Variation is measured as the squared distance from the points to the fitted line. Because no distributional assumptions are made, no confidence limits can be placed on the estimated line. The measure of the total variation is

$$\text{Total variation} = \sigma_{11}\sigma_{22} - \sigma_{12}^2 . \tag{7-16}$$

The fraction of the variation explained is derived from the eigenvalues of the covariance matrix, and the regression line corresponds to the first eigenvector. That is, the eigenvalues are the solution of

$$\left| \begin{pmatrix} \sigma_{11} & \sigma_{12} \\ \sigma_{12} & \sigma_{22} \end{pmatrix} - \lambda \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} \right| = 0 . \tag{7-17}$$

The values of λ which satisfy equation (7-17) are

$$\lambda = \frac{\sigma_{11} + \sigma_{22} \pm \sqrt{(\sigma_{11} - \sigma_{22})^2 + 4\sigma_{12}^2}}{2} . \tag{7-18}$$

The slope of the line corresponding to the largest eigenvalue, λ_1, is

$$\beta = \frac{-\sigma_{22} - \lambda_1}{\sigma_{12}} . \tag{7-19}$$

The intercept, β_0, is easily calculated because the line must pass through the mean of the data.

The measure, percent of variation explained, is a generalization of the multiple R^2 measure from a single dependent variable, but its behavior is somewhat different. For a two variable problem it can be calculated as $\lambda_1/(\lambda_1 + \lambda_2)$. In general, for correlations near 1, it will be about twice as good (.975 to .98 instead of .95), but for correlations near 0, the behavior is not as simple. As a result, it can only be used to compare one orthogonal regression with another. Because the standard correlation coefficient is a non-parametric measure of association, it can be used for orthogonal regression as well. The results of fitting by U.S. EPA of an orthogonal regression model to the previous example are in Table 7-33. The slope and intercept are almost identical to the normal error model values shown in Table 7-31.

TABLE 7-33. RESULTS OF AN ORTHOGONAL REGRESSION ANALYSIS OF THE EXPOSURE DATA FROM JAPAN

Y = intercept + slope X	
Variable	Beta
Intercept	10.83
Slope	0.475
Total variation	5686.9
Percent explained	98.5

Source of data: U.S. EPA reanalyses of data from Tamura et al. (1996).

Measurement Error Models

In general, most linear regression analyses assume the independent variable has no measurement error. When this error exists and no correction is made for it, the estimated regression coefficients tend to be biased towards zero. Because we often have multiple monitors we can often attempt to estimate these components of variation, and therefore correct our estimated regression coefficients. The solution usually requires some additional assumptions—in particular the assumption of multivariate normality is necessary for most of the solutions. Additionally, some information must be available about the error variance. Either the error variance of the independent variable or the dependent variable, or the ratio of the error variance to the variance of the dependent variable must be known exactly. In some cases, these values are

known with sufficient accuracy from other experiments so that the values can be treated as known.

Much of the material on measurement error in continuous variables comes from the work of Kendall and Stuart (1961) and Fuller (1987). Both authors make the same distinction that was made in the earlier section regarding the fixed or random nature of the independent variable. We will consider the more interesting case of measurement error in an independent random variable.

This subsection assumes a model with a continuous dependent variable and a continuous independent variable whose values are considered to be random and measured with error. For example, Hasabelnaby et al. (1989) described an analysis of pulmonary function data using measurements of NO_2 exposure as a covariate. The true NO_2 exposure was assumed to be a random variable which was estimated by sampling NO_2 levels in the home for two weeks out of the year. The other terms in the model were height and gender of the individual, and these were measured with little or no error.

The single random independent variable model assumes a single independent variable whose values, x_i, are random values. The model is

$$y_i = \beta_0 + \beta_1 x_i , \tag{7-20}$$

and we wish to estimate β_0 and β_1. Assume that the expected value of x is μ_x, the expected value of y is μ_y, and that the variance of x is σ_{xx}. We do not observe y_i and x_i, but rather Y_i and X_i, where

$$Y_i = y_i + \gamma_i \quad \text{and} \tag{7-21}$$

$$X_i = x_i + \delta_i , \tag{7-22}$$

and where γ_i is normal with mean 0 and variance σ_{yy} and δ_i is normal with mean 0 and variance σ_{xx}. The covariances between x_i, δ_i, and γ_i are assumed to be zero. This assumption implies that the vector (Y,X) is distributed as a bivariate normal vector with mean

$$E(Y,X) = (\mu_y, \mu_x) = \beta_0 + \beta_1 \mu_x, \mu_x \tag{7-23}$$

and covariance

$$\begin{bmatrix} \sigma_{YY} & \sigma_{XY} \\ \sigma_{XY} & \sigma_{XX} \end{bmatrix} = \begin{bmatrix} \beta_1^2 \sigma_{xx} + \sigma_{xx} & \beta_1 \sigma_{xx} \\ \beta_1 \sigma_{xx} & \sigma_{xx} + \sigma_{yy} \end{bmatrix}. \tag{7-24}$$

Let $\hat{\beta}_1$ be the standard regression estimate based on the observed data, (Y_i,X_i),

$$\hat{\beta}_1 = \left[\sum_{i=1}^{n} (X_i - \bar{X})^2\right]^{-1} \sum_{i=1}^{n} (X_i - \bar{X})(Y_i - \bar{Y}) . \tag{7-25}$$

The expected value of $\hat{\beta}_1$ is

$$E(\hat{\beta}_1) = \sigma_{XX}^{-1} \sigma_{XY} = \beta_1 (\sigma_{xx} + \sigma_{yy})^{-1} \sigma_{xx} . \tag{7-26}$$

Thus, for the bivariate normal model, the least squares regression coefficient is biased towards zero. The ratio, $\sigma_{XX}^{-1}\sigma_{xx}$ is known by several names including the attenuation, the reliability ratio, and in genetics as the heritability (Fuller, 1987).

Maximum likelihood equations can be set up for the bivariate normal model with measurement error. The first and second moments, which are sufficient to determine the distribution, will give five equations in the six unknown parameters, μ_x, σ_{XX}, σ_{xx}, σ_{yy}, β_0, and β_1. Clearly, some additional information is needed to make the problem identifiable. The three possibilities for additional information are σ_{xx}, σ_{yy}, or the ratio $\sigma_{xx}^{-1}\sigma_{yy}$, which lead to three different solutions. Two of these solutions are discussed in the following subsections.

If the measurement error in X, σ_{xx}, is known, then the solution is straightforward. For example, assume we know the variation between the ambient monitors because we have multiple monitors. Let S_{XX} be the maximum likelihood estimate of σ_{XX}, S_{YY} be the maximum likelihood estimate of σ_{YY}, and S_{XY} be the maximum likelihood estimate of σ_{XY}. The maximum likelihood estimate of β_1 becomes

$$\hat{\beta}_1 = S_{XY} / (S_{XX} - \sigma_{xx}) . \quad (7\text{-}27)$$

Note that this estimator reduces to equation (7-25) when the measurement error in x, σ_{xx}, is 0.

If the measurement error in Y, σ_{yy}, is known, then there is a comparable solution. Let S_{XX}, S_{YY}, and S_{XY} be defined as before. The maximum likelihood estimate of β_1 becomes

$$\hat{\beta}_1 = (S_{YY} - \sigma_{yy}) / S_{XY} . \quad (7\text{-}28)$$

All of this was based on the assumption that there was a true relationship between *x* and *y* that had no error. If, in fact, there was some error so that

$$y_i = \beta_0 + \beta_1 x_i + \varepsilon_i , \quad (7\text{-}20)$$

where ϵ_i is normal with mean 0 and variance σ_{ee}^2, then the estimate of β_1 would still come from equation (7-25), but the correlation would be estimated as

$$\hat{\rho} = \frac{\hat{\sigma}_{xy}}{\sqrt{\sigma_{xx}\,\sigma_{yy}}} = \frac{\hat{\sigma}_{xy}}{\sqrt{(S_{XX} - \hat{\sigma}_{xx})(S_{YY} - \hat{\sigma}_{yy})}} . \quad (7\text{-}30)$$

In order to estimate σ_{xx} and σ_{yy}, we can use an analysis as described in the following section. This correlation represents the upper bound to the observed correlation. That is, it is the correlation of the personal and ambient monitors if we had an infinite number of both. Under the assumption of equation (7-20), the value of this correlation is 1.

Components of Variance Models

If we have measurements from several individuals over time or several ambient monitors over time, then these measurements can be used in an analysis of variance (ANOVA) model. The purpose of the model is to estimate the variation between individuals and/or the variation between monitors. This information can then be used to adjust our slope estimates as described earlier, as well as letting us estimate the correlation between ambient and personal monitors assuming we had an infinite sample of both.

The logical analysis for this kind of data is a repeated measures design (see Winer, 1962, pp. 105-124). For most examples, the necessary components can be obtained from the results of a standard two-way ANOVA table. For example, consider the data of Tamura et al. (1996) after the missing values have been estimated (Tables 7-28, 7-29). There are 7 individuals measured over 11 48-h periods, resulting in the following ANOVA Table 7-34.

TABLE 7-34. RESULTS OF AN ANOVA ANALYSIS OF THE EXPOSURE DATA FROM JAPAN

Source of Variation	D.F.	S.S.	M.S.
date	10	9235.41	923.54
person	6	634.53	105.76
date × person	60	2248.66	37.48
Total	76	12118.60	

Source of data: U.S. EPA reanalyses of data from Tamura et al. (1996).

These results indicate that the mean square error for person is 105.76. This represents an estimate of $7\,\sigma_{yy} + \sigma_{ee}$ (mean squared error). The value, 37.48, represents an estimate of σ_{ee}, so that σ_{yy} can be estimated by (105.76 - 37.48) / 7 = 9.75. Because we will actually use the mean of 7 persons to estimate the average, the variance component we need for equation (7-28) is estimated by 9.75/7 = 1.39.

For example, consider the data of Tamura et al. (1996). From the above analysis, we have an estimate of the person variation, σ_{yy}, of 1.39 (for the mean of 7 individuals). Thus using equation (7-28), we can estimate β_1 as (119.97 - 1.39) / 232.83 = 0.509.

7.6.3 U.S. EPA Analysis of Data Sets

7.6.3.1 Tokyo, Japan Data Set

The data set of Tamura and Ando (1994) and Tamura et al. (1996) presents an interesting problem. Shown in Table 7-35 is the correlation matrix for average personal exposure with the two nearby ambient sites as well as their average. The Yamato site is located near a highway intersection 0.7 km from the central Itabashi site.

TABLE 7-35. COVARIANCE AND CORRELATION MATRIX FOR AVERAGE PERSONAL EXPOSURE AND AMBIENT EXPOSURES FROM JAPAN

Covariance/Correlation Matrix (Correlation below diagonal)

	Average Personal	Itabashi Site	Yamato Site	Average Site
Average person	119.97	232.83	308.81	270.82
Itabashi site	(0.951)	499.30	748.50	623.90
Yamato site	(0.736)	(0.874)	1467.62	1108.06
Average site	(0.840)	(0.949)	(0.983)	865.98

Source of data: U.S. EPA reanalyses of data from Tamura et al. (1996).

Note that the correlation of the average personal exposure is much higher with the Itabashi site than with the Yamato Site or the Average of the two sites. The estimated components of variance can give strange results when there are only two sites and one is much more highly correlated. For this reason, only the Itabashi site is used in the following analyses. If there had been additional sites it would have been possible to make all of the analyses in Table 7-36, but only those single site analyses are included at this time.

7.6.3.2 Phillipsburg, New Jersey Data Set

The personal exposure data (Lioy et al., 1990) contained some missing values and three outlier values, and they all were estimated as described earlier. The results of U.S. EPA reanalyses are shown in Table 7-37. In order to estimate the error variances, these data were used in an analysis of variance as described earlier. The results are shown in Table 7-38.

TABLE 7-36. SUMMARY OF RESULTS OF THE ANALYSIS OF THE EXPOSURE DATA FROM JAPAN

Regression Model	β_1	β_0
Linear, normal error	0.466	11.3
Linear, lognormal error	0.431	13.1
Orthogonal	0.475	10.8
Linear adjusted for person error	0.509	8.9
Linear adjusted for ambient error	(Not available)	
Measures of Association		**Value**
Correlation of personal averages with Itabashi site		0.951
Correlation adjusted for measurement error		(Not available)
Average correlation of ambient with mean person		(Not available)
Average correlation of person with mean ambient		0.872
Fraction of variation explained by orthogonal regression		0.985

Source: U.S. EPA reanalyses of data from Tamura et al. (1996).

The site monitoring data contained some missing values, and they were estimated by U.S. EPA as described in Section 7.6.2.1. The means, covariances and correlations were also estimated. The results are in Table 7-39. In order to estimate the error variances, the same data were used in an analysis of variance as described earlier. The results of the EPA analyses are shown in Table 7-40. The individual exposure values were averaged as well as the site exposure values. These means are shown in Table 7-41.

The same regression analyses described earlier were performed by U.S. EPA. A plot of the linear regression is shown in Figure 7-32. The orthogonal regression gives virtually an identical plot and is not shown. The results of the analyses are in Table 7-42.

Note that all estimated regression equations are quite similar. The interesting value is the correlation adjusted for measurement error. This represents an estimate of the correlation between the mean of an infinite number of personal samplers and the mean of an infinite number of fixed site samplers. This value is relatively close to one, but we do not have good estimates of its variance to tell if the value is really different from one.

TABLE 7-37. PERSONAL EXPOSURE SUSPENDED PARTICULATE MATTER DATA FROM PHILLIPSBURG, NEW JERSEY. MISSING VALUES ESTIMATED ( ); OUTLIER VALUES RECOMPUTED [ ].

	Person Identifier ($\mu g/m^3$)													
Day	01	02	11	31	41	42	51	52	61	62	81	82	91	92
1	59	85	54	39	(53.2)	36	41	28	123	67	96	79	50	32
2	52	58	85	17	(76.7)	45	50	53	104	56	50	49	66	63
3	74	69	94	56	86	77	90	93	200	134	166	81	77	187
4	115	88	136	104	65	116	112	120	125	272	193	98	164	172
5	65	37	139	38	77	64	56	52	184	190	79	49	(95.7)	89
6	45	16	56	22	34	27	28	21	60	58	57	12	54	99
7	75	77	65	35	36	80	27	34	92	(110.2)	124	77	107	184
8	104	81	79	67	83	32	69	61	112	91	144	69	96	198
9	84	29	48	56	85	122	30	36	57	96	156	123	91	[100.6]
10	55	29	70	35	59	81	25	39	199	77	63	41	66	135
11	10	60	65	25	36	[48.1	49.4]	43	93	84	99	32	78	122
12	39	59	80	23	127	57	32	35	121	95	31	45	63	72
13	26	44	65	35	31	47	114	67	47	95	71	18	31	109
14	45	44	89	17	105	117	(24.8)	24	117	63	44	14	57	108

Source: Data from Lioy et al. (1990). Missing values estimates and recomputed outlier values calculated by U.S. EPA.

TABLE 7-38. RESULTS OF AN ANOVA ANALYSIS OF THE PERSONAL EXPOSURE DATA OF PHILLIPSBURG, NEW JERSEY

Source of Variation	d.f.	s.s.	m.s.
Date	13	119,600	9202
Person	13	103,300	7942
Date × Person	169	149,900	887
Total	195	372,800	

Source: U.S. EPA reanalyses of data from Lioy et al. (1990).

TABLE 7-39. SAM SITE CONCENTRATIONS, PM_{10} DATA ($\mu g/m^3$) FROM PHILLIPSBURG, NEW JERSEY [Missing Values Estimated ( )].

Day	Site 101	Site 102	Site 103	Site 020
01	26	41	28	24
02	51	(55.6)	55	46
03	94	(101.8)	112	98
04	148	155	165	209
05	76	81	76	85
06	15	17	13	50
07	44	47	49	51
08	101	105	119	99
09	59	67	68	66
10	46	52	50	57
11	37	36	35	34
12	28	33	28	28
13	27	27	27	25
14	21	23	19	38
Means	55.2	60.1	60.3	65.0
Covariance/Correlation Matrix (Correlation below diagonal)				
Site 101	1313.9	1346.5	1538.9	1596.6
Site 102	0.995	1393.8	1581.4	1630.9
Site 103	0.996	0.994	1816.2	1850.1
Site 020	0.943	0.935	0.929	2183.4

Source: U.S. EPA reanalyses of data from Lioy et al. (1990).

TABLE 7-40. RESULTS OF AN ANOVA ANALYSIS OF THE SITE EXPOSURE DATA OF PHILLIPSBURG, NEW JERSEY

Source of Variation	d.f.	s.s.	m.s.
Site	3	671	223.6
Day	13	90286	6945.1
Site × Day	39	3615	92.7
Total	55	94572	

Source: U.S. EPA reanalyses of data from Lioy et al. (1990).

TABLE 7-41. AVERAGE PERSONAL PM_{10} EXPOSURE DATA COMPARED WITH THE SITE EXPOSURE DATA FOR PHILLIPSBURG, NEW JERSEY

Day	Ambient Average ($\mu g/m^3$)	Average Personal ($\mu g/m^3$)
1	29.75	60.15
2	51.55	58.91
3	101.45	106
4	169.25	134.29
5	79.5	86.76
6	23.75	42.07
7	47.75	80.23
8	106	91.86
9	65	79.19
10	51.25	69.57
11	35.5	60.74
12	29.25	62.79
13	26.5	57.14
14	25.25	62.04

Source: U.S. EPA reanalyses of data from Lioy et al. (1990).

7.6.3.3 Beijing, China Data Set

The Beijing, China data set reported by the World Health Organization (1985) is listed in Table 7-43. From these data, daily mean values of the ambient and personal exposure values were computed. An U.S. EPA reanalysis of these data is shown in Table 7-44 and in Figure 7-33. The results of the analysis indicate that there is not a significant linear relationship between the personal and ambient monitoring data. For this reason, it does not

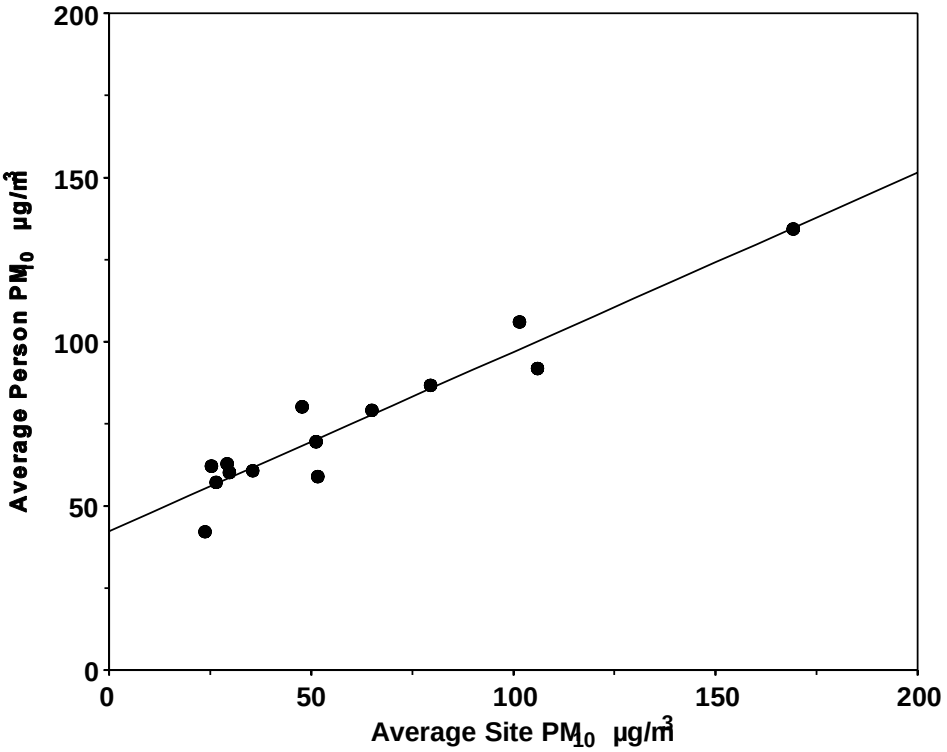

Figure 7-32. Plot of relationship between average personal PM_{10} exposure versus ambient PM_{10} monitoring data from Phillipsburg, NJ and regression line calculated by U.S. EPA.

Source: Lioy et al. (1990).

make any sense to adjust the coefficient for measurement error. The subjects all worked at the same institute so their daytime personal exposures may not have been independent of each other.

7.6.3.4 Riverside, California Data Set

Both the personal exposure and the monitoring data used in analyses by Clayton et al. (1993) contained some missing values, and they were estimated by U.S. EPA as described earlier. The estimated correlation/covariance matrix results of U.S. EPA reanalyses of these data are shown in Table 7-45.

Because the individual monitors were placed on different individuals each period, we can't really estimate the variation between individuals. Based on previous analyses, we know that most of the residual is variation between individuals, and so we will use this as a

TABLE 7-42. RESULTS OF THE ANALYSIS OF THE EXPOSURE DATA FROM PHILLIPSBURG, NEW JERSEY

Regression Model	β_1	β_0
Linear, normal error	0.546	42.3
Linear, lognormal error	0.560	41.4
Orthogonal	0.556	41.9
Linear adjusted for person error	0.556	41.9
Linear adjusted for ambient error	0.587	40.1
Measures of Association		**Value**
Correlation of averages		0.955
Correlation adjusted for measurement error		0.974
Average correlation of ambient with mean person		0.944
Average correlation of person with mean site		0.633
Fraction of variation explained by orthogonal regr.		0.984

Source: U.S. EPA reanalyses of data from Lioy et al. (1990).

surrogate. On average there were 3.5 persons per period and this number of individuals was used in the analysis of variance shown in Table 7-46. The dichot monitoring data contained little missing data, and so it was analyzed against the personal monitoring data for those days with data. The results of the linear regression are in Table 7-47 and are shown graphically in Figure 7-34. The individual exposure values were averaged so that they could be compared with the site exposure values. These means are shown in Table 7-48. Note that the orthogonal regression slope is larger than either of the linear regression slopes. Note also that the linear regression slope adjusted for measurement error is larger than any of the other slopes.

7.6.3.5 Azusa, CA Data Set

The Azusa, CA data set for PM_{10} reported on by Wiener et al. (1990) was described earlier in Section 7.4.1.1.1 and presented in Table 7-21a. The same regression analyses described earlier in this section were performed on the 24-h cross-sectional data and the results are shown in Table 7-49. A plot of the linear regression analysis, resulting in a

TABLE 7-43. PERSONAL AND AMBIENT EXPOSURE DATA FOR BEIJING, CHINA (mg/m^3)

Day	Personal	Ambient	Day	Personal	Ambient
1	0.13	0.19	6	0.15	0.42
2	0.15	0.25	6	0.17	0.42
2	0.10	0.25	6	0.13	0.42
2	0.12	0.25	6	0.16	0.42
2	0.23	0.25	6	0.21	0.42
2	0.14	0.25	6	0.08	0.42
3	0.11	0.31	7	0.35	0.44
3	0.09	0.31	7	0.24	0.44
3	0.09	0.31	7	0.20	0.44
4	0.31	0.33	8	0.15	0.53
4	0.12	0.33	9	0.23	0.55
4	0.13	0.33	9	0.18	0.55
4	0.35*	0.33	9	0.10	0.55
4	0.12	0.33	9	0.38	0.55
4	0.25	0.33	10	0.11	0.59
5	0.10	0.36	11	0.23	0.69
5	0.22	0.36	11	0.32	0.69
5	0.32	0.36	11	0.11	0.69
5	0.12	0.36	11	0.21	0.69
5	0.08	0.36	11	0.11	0.69
5	0.13	0.36	11	0.20	0.69
5	0.07	0.36	11	0.29	0.69

*The only personal value higher than the ambient value.

Source: World Health Organization (1985).

TABLE 7-44. RESULTS OF LINEAR REGRESSION ANALYSIS FOR THE BEIJING, CHINA EXPOSURE DATA

Linear regression analysis of average personal exposure versus ambient exposure

Y = intercept + slope X

Variable	Beta	Std. Error Beta
Intercept	0.116	0.040
Slope	0.142	0.088

ANOVA Table

Source	Sum of Squares	Mean Square Error	D.F.	F-Value
Regression	0.0179	0.00893	2	1.2911
Error	0.2835	0.00692	41	
TOTAL	0.3014	0.00701	43	

R-squared = 0.05925, r = 0.2434

Log-likelihood = −46.95

Source: U.S. EPA reanalyses of data from World Health Organization (1985).

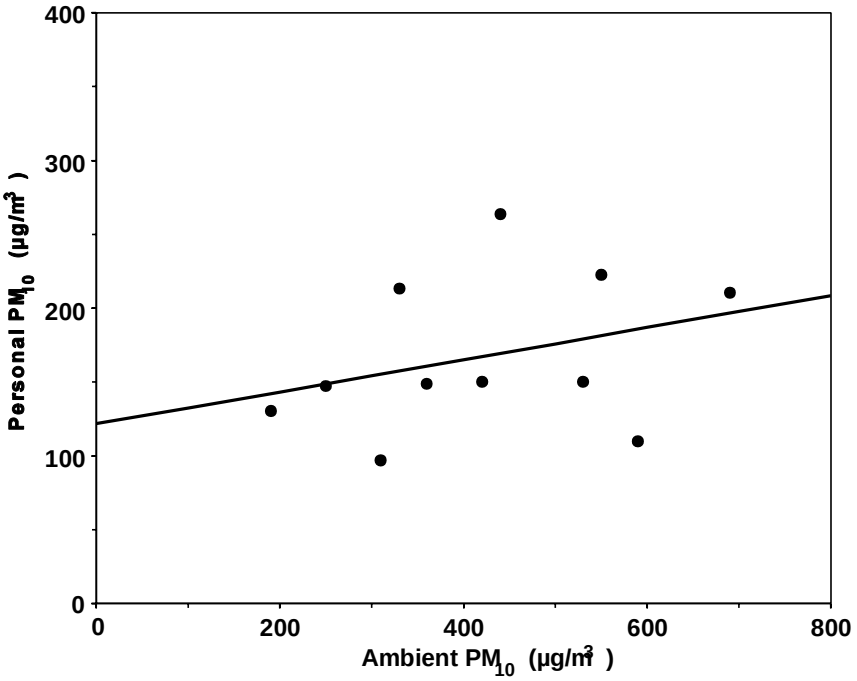

Figure 7-33. Plot of means of personal exposures and ambient PM_{10} from Beijing, China and regression line calculated by U.S. EPA.

Source: U.S. EPA reanalyses of data from World Health Organization (1985).

TABLE 7-45. ESTIMATED MEAN VECTOR, COVARIANCE MATRIX, AND CORRELATION MATRIX OF PERSONAL EXPOSURE PM_{10} DATA FROM RIVERSIDE, CALIFORNIA (24-h, $\mu g/m^3$)

	Monitor					
	Personal	Indoor	Backyard	Dichot	Wedding	PEM-SAM
Means	109.9	79.9	91.7	71.2	68.4	80.4
Covariance/Correlation Matrix (Correlation below diagonal)						
Personal	1055.0	917.4	1024.7	749.0	838.9	913.7
Indoor	(0.849)	1107.6	1017.9	832.7	897.0	987.4
Backyard	(0.725)	(0.703)	1893.2	1165.6	1296.9	1427.4
Dichot	(0.707)	(0.767)	(0.821)	1063.4	1116.6	1232.9
Wedding	(0.721)	(0.753)	(0.832)	(0.956)	1282.8	1337.1
PEM-SAM	(0.736)	(0.776)	(0.858)	(0.989)	(0.976)	1462.3

Source: U.S. EPA reanalyses of data reported on by Pellizzari et al. (1992).

TABLE 7-46. RESULTS OF AN ANOVA ANALYSIS OF THE PERSONAL EXPOSURE DATA OF RIVERSIDE, CALIFORNIA

Source of Variation	D.F.	S.S.	M.S.
period	46	167,400	3640
residual	114	275,000	2412
Total	160	442,400	

Source: U.S. EPA reanalyses of data reported on by Pellizzari et al. (1992).

TABLE 7-47. RESULTS OF THE ANALYSIS OF THE EXPOSURE DATA FROM RIVERSIDE, CALIFORNIA

Regression Model	β_1	β_0
Linear, normal error	0.6174	59.7
Linear, lognormal error	0.6185	57.1
Orthogonal	0.8071	44.2
Linear adjusted for person error	0.9675	31.0
Linear adjusted for ambient error	(Not applicable)	

Measures of Association	Value
Correlation of averages	0.721
Correlation adjusted for measurement error	(Not applicable)
Fraction of variation explained by orthogonal regr.	0.864

Source: U.S. EPA reanalyses of data reported on by Pellizzari et al. (1992).

negative slope, is shown in Figure 7-35. There clearly is no relationship between the pooled PEM and SAM variables for this data set. The statistical explanation for the negative correlation and slope (PEM decreases with increasing SAM) is that one of the observations (PEM = 273 $\mu g/m^3$, SAM = 48 $\mu g/m^3$, for House 9, Day 10, person 1, as shown in Table 7-21a) is an outlier (273 $\mu g/m^3$ > mean + 3*SD). Removal of this single datum point changes both the correlation and the slope to slightly positive values of similar magnitude. Because of the insignificance of the slope and correlation, further adjustments for measurement error do not make sense.

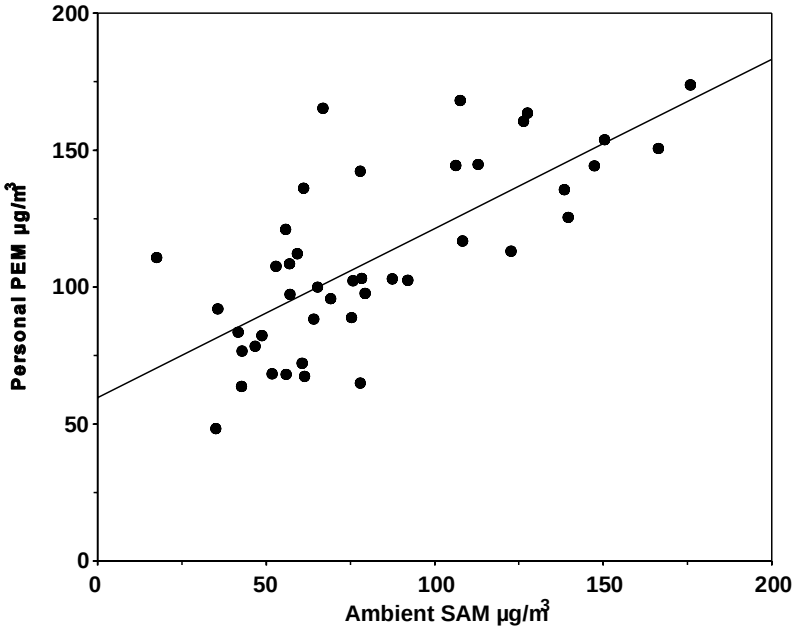

Figure 7-34. PTEAM mean 24-h PM_{10} data compared for personal PEM and SAM.

Source: U.S. EPA reanalyses of data reported on by Pellizzari et al. (1992).

7.6.4 Discussion of Statistical Analyses: Mean PEM Versus Mean SAM

The Beijing study had an insignificant positive slope and the Azusa study gave an estimated slope less than zero that becomes insignificant positive with the removal of one outlier. Possible explanations for the low slope of the Beijing study may be related to the unusually low ratio of PEM to SAM of order 0.4. Either the SAM $PM_{3.5}$ monitor that was used may have been influenced by a local PM source, and thereby was not representative of the Beijing locality where the subjects worked and lived, or the air exchange between indoors and outdoors during the winter period was greatly minimized for personal comfort.

In the Beijing dataset of 44 pairs of simultaneous SIM and SAM (Table 7-43) only one $PM_{3.5}$ PEM value was greater than SAM, as opposed to Azusa where in the 50 pairs of simultaneous SIM and SAM (Table 7-21b) only six $PM_{2.5}$ PEM values were less than SAM. On a day where SAM $PM_{3.5}$ reached 690 µg/m^3 in Beijing, seven simultaneous PEM values all ranged between 110 µg/m^3 and 320 µg/m^3. In relation to Figure 7-16, these PEM/SAM ratios between 0.16 and 0.45 correspond to low air exchange rates of order 0.1 to 0.3 air changes per hour. In the tightly-sealed poorly-heated building where all the subjects worked

TABLE 7-48. AVERAGE 24-HOUR PM_{10} PERSONAL EXPOSURE DATA COMPARED WITH THE PEM-SAM SITE EXPOSURE DATA FOR RIVERSIDE, CALIFORNIA ($\mu g/m^3$)

Period	Average Personal	PEM-SAM Site
1	48.3	35.1
3	83.6	41.7
5	108.6	56.9
7	88.3	64.1
9	68.3	51.7
11	121.0	55.8
15	68.2	56.0
17	95.8	69.1
19	102.5	92.0
21	116.8	108.2
23	160.5	126.4
25	97.7	79.4
27	72.2	60.7
29	107.6	52.9
31	103.0	87.4
37	165.3	66.8
39	144.4	106.2
41	135.6	138.5
43	168.2	107.5
47	173.8	175.9
49	144.9	112.9
51	65.0	77.9
53	76.7	42.8
57	110.9	17.6
59	78.4	46.7
61	136.1	61.1
63	103.1	78.4
65	142.4	77.9
67	163.6	127.6
69	153.7	150.4
71	144.2	147.4
73	150.6	166.4
75	125.4	139.6
77	112.1	59.2
79	63.7	42.7
81	67.5	61.4
83	102.2	75.8
85	92.0	35.7
87	100.0	65.3
89	88.9	75.3
91	113.0	122.7
93	82.4	48.8
95	97.3	57.1

Source: U.S. EPA-calculated 24-h averages, based on 12-h data reported on by Pellizzari et al. (1992).

TABLE 7-49. RESULTS OF THE LINEAR REGRESSION ANALYSIS OF THE EXPOSURE DATA FROM AZUSA, CALIFORNIA

Variable	Beta	Std. Error Beta
Intercept	119.1	13.77
Slope	−0.054	0.201
Covariance Matrix of Parameter Estimates		Slope
	Intercept	
Intercept	189.7	−2.543
Slope	−2.543	0.040
Log-likelihood = −263.4		

ANOVA Table

Source	Sum of Squares	Mean Square Error	D.F.	F-Value
Regression	111.2	55.6	2	0.0363
Error	76590	1531.8	50	
TOTAL	76700	1475.1	52	
R-squared = 0.0015				

Source: U.S. EPA reanalyses of data reported on by Wiener et al. (1990).

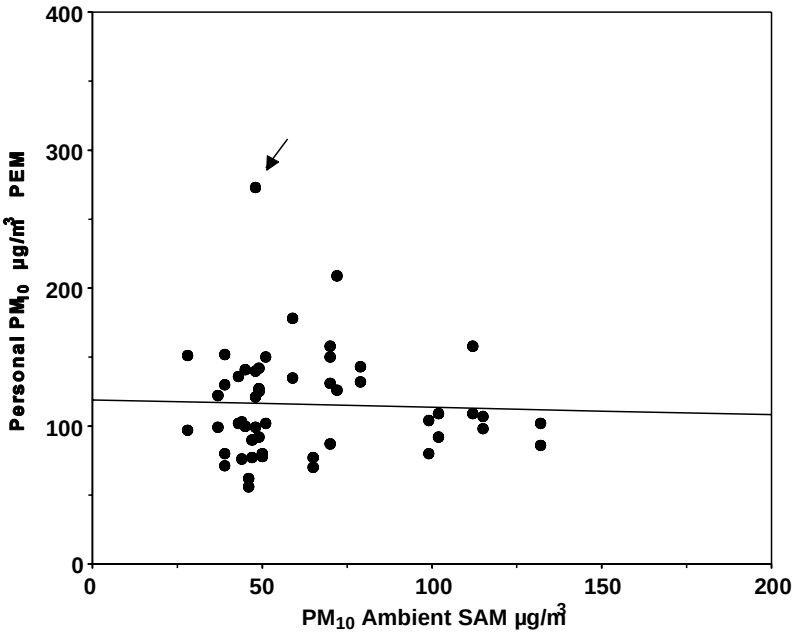

Figure 7-35. Plot of ambient and personal monitoring PM_{10} data from Azusa, CA and calculated (slightly negative slope) regression line, which becomes positive if single outlier value (↙) is deleted.

Source: U.S. EPA reanalyses of data reported on by Wiener et al. (1990).

during the Beijing winter, a small variation in air exchange could result in a relatively large difference in the indoor PM, which would result in PEM that appears to be uncorrelated with SAM. If a contribution of PM generated by personal activity and ETS is subtracted from the PEM values then the estimated air exchange rates would be even lower. The remaining discussion will be based on the other three studies, realizing that the discussion is not supported by these two studies.

The major conclusions which can be reached from the remaining three studies are as follows.

(1) The average of several ambient monitors correlates better with mean personal exposure than does an individual site (as would be predicted by the Central Limit Theorem).

(2) The average of several personal monitors correlates better with mean ambient exposure than does the ensemble of individual monitors.

(3) There is no evidence of the existence of a maximum (ceiling) correlation between personal and ambient measurements. The only study with fixed multiple ($n > 2$) ambient SAM locations and multiple personal monitors is the Phillipsburg, NJ, study. The estimated correlation adjusted for measurement error was 0.97. The true (unknown) correlation between an infinite average of personal monitors with an infinite average of fixed site monitors may be different (smaller) in other locations, but we do not have the data to evaluate that.

(4) The correlation coefficient is probably the best measure of association between personal and ambient measurements. It can be used independent of the regression technique or model and does not assume a distributional form. The "percent of variation explained" as derived from orthogonal regression is not comparable to any measure used for other models.

(5) The choice of a model (linear, linear with lognormal error, orthogonal) makes less difference than the adjustment for measurement error.

(6) Based on the results of the Phillipsburg, NJ, analysis, one or more fixed site monitors can do an excellent job of predicting the average of all personal exposures (if they could be measured) even though the prediction for most individual exposures is quite poor. This is also supported by the Tokyo, Japan, data set (Tamura et al., 1996). The other data sets did not provide adequate information to either confirm or deny this conclusion.

The value of the improvement of the mean PEM relationship to SAM is that it provides a better visualization that helps in understanding how mean PEM varies with SAM. It thus provides a measure of the validity of the use of a daily PM SAM as a surrogate for the mean PM

PEM in the community for nonsmokers. It is clear that the uncertainty in predicting mean personal exposure PM is much smaller than the uncertainty in predicting the personal exposure PM for a nonsmoking individual when we note that the means have a much smaller variability about the line as shown in Figures 7-31, 7-32, and 7-34.

There appears to be two distinct categories of cross-sectional exposure studies that were examined: In the first type of study, such as Lioy et al. (1990), Clayton et al. (1993), and Tamura et al. (1996), there is a significant R^2 between individual PM PEM and PM SAM. In this category, there is an appreciable improvement in correlation between the mean PEM and SAM. It has been suggested that these cases with higher correlation of PEM PM with SAM PM may arise where the fine portion of the ambient PM ($PM_{2.5}$) is highly variable from day-to-day, and the ambient coarse fraction is relatively constant (Wilson and Suh, 1995). In an urban area, the fine particle composition and the fine particle concentration are often highly correlated from site-to-site on any given day. This is due, in part, to the gas phase reactions of SO_x and NO_x, associated with regional sources, to produce sulfates and nitrates in the submicron range. Because of the long residence times of these species due to their negligible deposition velocities, they are well mixed throughout the air mass (Suh et al., 1995; Burton et al., 1996).

On the other hand, ambient coarse particles are generated locally, and they have higher deposition velocities than the fine particles. Their impact may then be limited by fallout to a locality downwind of their emission point, as they are not readily transported across an urban area. Therefore, during an air pollution episode, people living in an urban area may be exposed to fine PM of similar chemical composition and concentrations, whereas they will be exposed to coarse PM of ambient origin with a chemical composition that can depend on the location of the exposure. Because ambient PM penetrates readily into a nonambient setting, the correlation between the mean $PM_{2.5}$ PEM and $PM_{2.5}$ SAM would be high because all the people would have similar exposure to the ambient fine PM - plus exposure to indoor generated $PM_{2.5}$ which may have less fluctuation in the absence of smoking.

In the second type of study, such as Sexton et al. (1984), Spengler et al. (1985), and Wiener et al. (1990), there is negligible correlation between individual PEM PM and SAM PM, and consequently there will be little correlation between their mean PEM and the SAM. In these cases, if the fine fraction is not an appreciable portion of the ambient PM, or there are significant

indoor sources, then the correlations between mean PM PEM and PM SAM will be lower and possibly not significantly different from zero.

7.7 IMPLICATIONS FOR PARTICULATE MATTER AND MORTALITY MODELING

PM related mortality may be specific to the most highly susceptible portion of the population. Such a cohort may be the elderly people with the most serious chronic obstructive pulmonary disease (COPD) and cardiac insufficiency. Smithard (1954) relates the findings of Dr. Arthur Davies (Lewisham coroner) who autopsied 44 people who died suddenly during the 1952 London Fog:

> "The great majority of deaths occurred in people who had pre-existing heart and lung trouble, that is to say they were chronic bronchitic and emphysematous people with consequent commencing myocardial damage. The suddenness of the deaths, Dr. Davies thought, was due to a combination of anoxia and myocardial degeneration resulting in acute right ventricular dilatation."

Mage and Buckley (1995) hypothesized that these people with compromised cardio-pulmonary systems may be relatively inactive, while selecting to live in homes or institutional settings without sources of indoor pollution. When their time is spent in clean settings (e.g. where smoking is prohibited), they would have little exposure to PM other than from the ambient pollution that intrudes into their living quarters (Sheldon et al., 1988a,b). The exposure to PM of this cohort, would be highly correlated with PM SAM, and so would be their mortality, if this ambient PM was reactive in their pulmonary tracts as described by West (1982). However, there have been no results reported of an exposure study done on people with COPD who correspond to the Lewisham mortality cohort. The cohort of five elderly housewives and two male retirees in Tokyo (Tamura et al., 1996) may come close to this susceptible cohort. Individual PM PEM of people outside these cohorts, who could be relatively insensitive to ambient PM, might not be significantly correlated with PM SAM, as reported in most of the other studies of nonsmokers cited in Table 7-26. This suggests a model to relate PM and mortality as follows. Let any person (j) on a given day have a probability of mortality, $p(m) = k_j X_j$, where k_j is the unit probability of mortality per $\mu g/m^3$ of PM per day, X_j is the daily average exposure to PM, $\mu g/m^3$, independent of k_j. Let us assume that each individual (j) has their own

personal value of k_j that can vary from day-to-day with changes in their respiratory health, such as a transient pulmonary infection (West, 1982).

The expectation of total mortality (M) in a community of size N can be shown to be the summation of k X over all individuals (j = 1 to N) as follows:

$$M = \Sigma\, k_j\, X_j \tag{7-31}$$

If k_j is independent of X_j, then we can define K as (1/N) Σ k_j, and the mean community exposure $\bar{X}$ as (1/N) Σ X_j, and it follows

$$M = N K \bar{X} \tag{7-32}$$

This implies that, given a linear relationship of mortality with PM PEM exposure (X) as assumed in most studies discussed in Chapter 12, the expected mortality is proportional to the mean community personal exposure to PM. The individual in the community, on any given day, with the highest probability of dying from a PM exposure related condition is that individual with the highest product $k_j X_j$, not necessarily the highest exposed individual with the maximum value of X_j (West, 1982).

The Phillipsburg, NJ, data set is a case in point. In this study, three subjects had excessively high PEM PM. These values were caused by a hobby involving welding in a detached garage (971 $\mu g/m^3$), a home remodeling activity (809 $\mu g/m^3$) and usage of an unvented kerosene heater (453 $\mu g/m^3$). Excessive PM generating activities are not expected of elderly people who may have compromised pulmonary systems. In fact, the elderly and infirm husband of the remodeler had a personal exposure of 45 $\mu g/m^3$ on the day of the remodeling activity. The indoor monitors in the homes of the welder and remodeler only recorded 55 $\mu g/m^3$ and 19 $\mu g/m^3$, respectively, during those events, indicating the specificity of the high exposure to only the individual involved. These three outliers were removed from the analysis and were replaced by the procedure for missing data of section 7.6.2.1, which estimates their exposures as if they had not done those specific activities responsible for their noncharacteristic exposures (see Table 7-37). This procedure is reasonable, since it is unlikely that these activities would be performed by individuals with pulmonary conditions similar to those of the Lewisham mortality cohort

(Smithard, 1954). As shown on Table 7-42 and Figure 7-32, the regression improves markedly to a value of $R^2 = 0.914$.

It is this relation of the average PM PEM exposure to PM SAM concentration, as shown in Figure 7-32 that may be a better representation of the true situation underlying the PM vs mortality relationships because of the "healthy worker" effect. Chronically ill people who are sensitive to PM might change their behavior to minimize their exposure to irritants. Consequently, healthy people with high PEM PM measures in occupations and indoor settings can cause the regression R^2 between PEM and SAM for nonsmokers to be low, but they may not be the individuals at highest risk of the acute effects of PM exposure.

7.7.1 Relative Toxicity of Ambient Particulate Matter and Indoor Particulate Matter

In the previous sections the SAM PM was evaluated as a predictor of PEM PM of nonsmokers on the implied basis that the health effects of PM were only mass dependent, and independent of chemical composition. It was shown in Table 7-26 that many early PM studies of PEM had a low correlation between PEM and SAM on a cross-sectional basis that was often not significantly different from zero. But, in the later studies (Tamura et al., 1996; Lioy et al., 1990), a significant relationship was observed between PEM and SAM on an individual basis. Further analysis showed that on a daily basis, SAM would appear to be a good predictor of mean community exposure to ambient PM_{10} of nonsmoke exposed people from the results of the Tokyo, Japan; Riverside, CA; and Phillipsburg, NJ; studies. However, there can be a large difference in toxicity of PM per unit mass which is related to the chemical composition, solubility and size of the particles. For example, mercury (Hg) and arsenic (As) have significantly different toxicities in their inorganic and organic forms. Hexavalent chromium (Cr) is more toxic than trivalent Cr. Anthropogenic PM, from combustion of fossil fuels, is much more toxic than PM of natural origin (Beck and Brain, 1982; Mage et al., 1996). Fine urban particulate matter generated by coal smoke during the 1952 London Fog at concentrations of order 2,000 $\mu g/m^3$ caused thousands to die (Holland et al., 1979; United Kingdom Ministry of Health, 1954) but 2,000 $\mu g/m^3$ of soil dust from dust storms (Hansen et al., 1993) would not have been as deadly.

Soil constituents that are tracked-in to a home on shoes, and are subsequently resuspended, contribute to the personal cloud (Roberts et al., 1990; Thatcher and Layton, 1995). "Even if this crustal PM is relatively inert, its presence in the lung potentiates the toxicity of the anthropogenic particles because it increases the residence time of the more toxic PM (WHO, 1995)" (Mage et al., 1996). This increase in soil constituents was also shown in the PTEAM study (Özkaynak et al., 1996) on Figure 7-22 "by observation that nearly all [soil] elements were elevated in personal samples" but sulfur, which is in the ambient fine mode, was not a personal cloud constituent. This is consistent with the observations of Wilmoth et al. (1991) that "extremely small particles (below two micrometers) require local airflow (sampling) velocities near 100 miles per hour [45 m/s] to overcome surface attraction forces and dislodge [them] for sampling".

Figure 7-36 shows an example of resuspension of Pb in a Denver, CO, home (Moschandreas et al., 1979). During the one-week sample, a wind shift brought a clean air mass to below 0.01 $\mu g/m^3$. In this time period, the average indoor Pb dropped from 0.085 to 0.048 $\mu g/m^3$. The residual 0.048 $\mu g/m^3$ represents the effect of resuspension by human activity. When the wind shifted again, and ambient Pb rose to 0.360 $\mu g/m^3$ the indoor Pb rose to 0.180 $\mu g/m^3$. Note the peaks in the indoor concentration of Pb up to and above 0.10 $\mu g/m^3$ during the clean air period which are indicative of variations in resuspension by human activities.

There is also some indication in laboratory animal studies, using transpleural catheterization and intratracheal instillation, that products of fossil fuel combustion are more acutely toxic to animals than wood smoke and soil constituents (U.S. Environmental Protection Agency, 1982, Table 12-6; Beck and Brain, 1982). Although these laboratory animal studies may have no direct relation to toxicity in humans, they provide an indication of their relative toxicity in animals when administered by those two routes.

In summary, there is evidence that not all PM constituents have the same toxicity per unit mass. These differences are due to differences in aerodynamic diameter and chemical composition. As shown on a Venn diagram (Figure 7-37, Mage [1985]), the focusing of the description of a PM_{10} exposure increases the ability to estimate the potential toxicity of the exposure. In the sequential description given below, the uncertainty in the toxicity of the mixture is decreased as more information is provided.

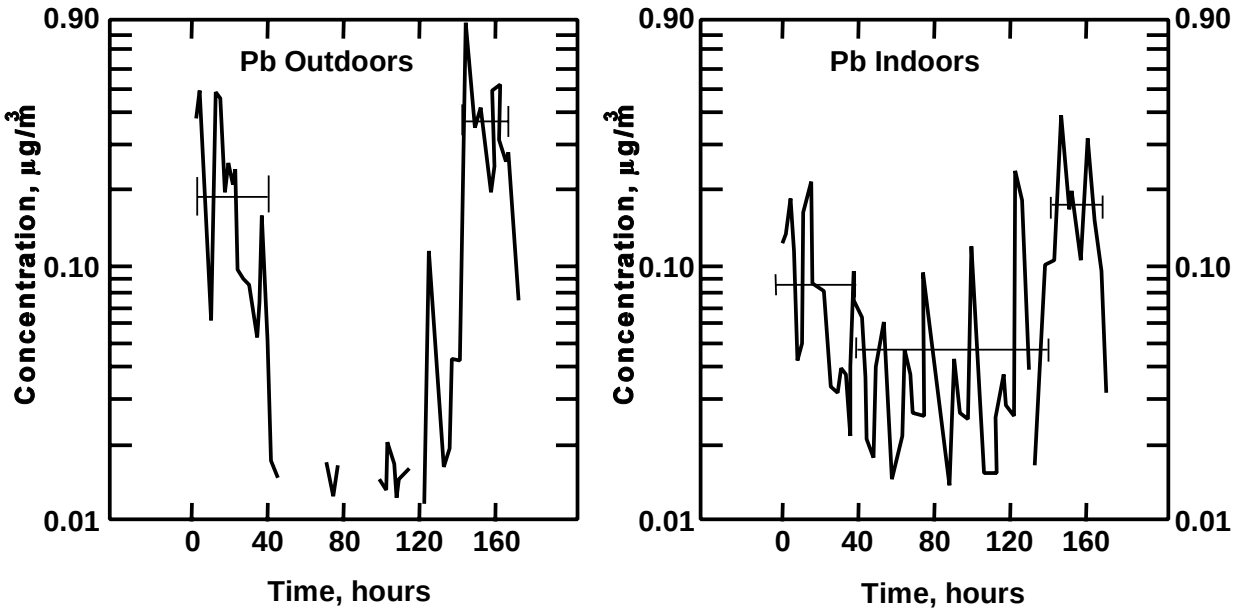

Figure 7-36. Comparison of indoor and outdoor concentrations of lead in a home in Denver, October 1976, for 1 week, starting at 1600 h. Mean values are given by horizontal bars.

Source: Moschandreas et al. (1979).

1. 2 µg/m^3 of PM_{10}.
2. 2 µg/m^3 of PM_{10} in the size interval 2 to 2.5 µm.
3. 2 µg/m^3 of PM_{10} in the size interval 2 to 2.5 µm, 50% of automotive origin and 50% of indoor source origin.
4. 2 µg/m^3 of PM_{10} in the size interval 2 to 2.5 µm, 50% of automotive origin and 50% of indoor source origin, 0.5 µg/m^3 of Pb, 0.5 µg/m^3 of BaP and 1 µg/m^3 of unspecified inorganic material.

As applied to human exposure to PM, this concept of differential toxicity suggests that data collections might benefit by providing data that would allow the toxicity of a PM exposure to be evaluated in terms of chemical information, in addition to the mass collected per unit volume.

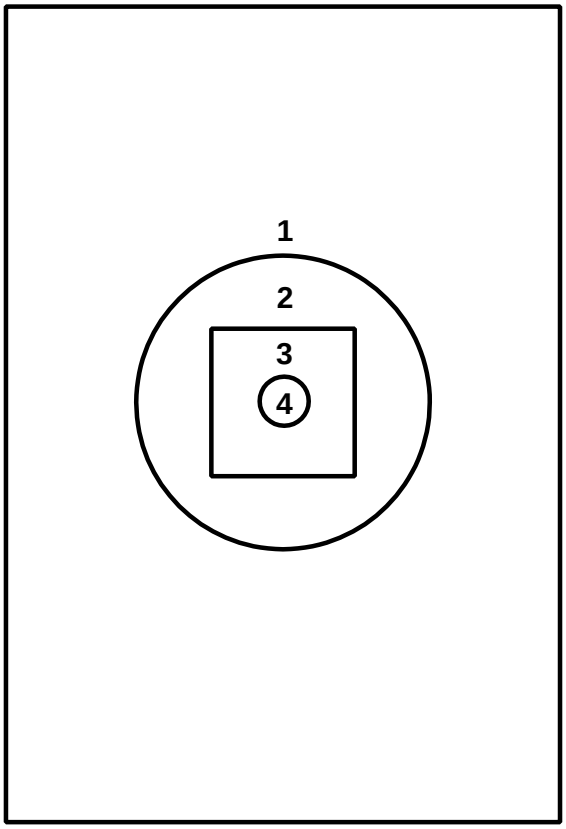

Figure 7-37. Venn diagram (Mage, 1985) showing focusing of information to more completely specify toxicity of a given PM mixture: (1) universe of all possible mixtures of PM with concentration of 2 $\mu g/m^3$; (2) subuniverse of all combinations of PM with concentration of 2 $\mu g/m^3$ in size interval 2.0 to 2.5 μm; (3) subuniverse of all combinations of PM with concentration of 2 $\mu g/m^3$ in size interval 2.0 to 2.5 μm AD with 50% of automotive origin and 50% from indoor sources; and (4) subuniverse of all combinations of PM with concentration of 2 $\mu g/m^3$ in size interval 2.0 to 2.5 μm AD with 50% of automotive origin and 50% from indoor sources; 25% Pb, 25% BaP and 50% unspecified inorganic materials.

7.7.2 Summary: Linkage of Ambient Concentrations of Particulate Matter to Personal Exposures to Particulate Matter

As described by Wilson and Suh (1995), total exposure to ambient PM (X_{ae}) of any given size range is equal to the summation of exposures to ambient PM over both ambient (X_a) and nonambient (X_{na}) microenvironmental conditions. Total exposure to PM is equal to X_{ae} plus exposure to nonambient PM concentrations generated independently of personal activities (X_{nai}) and nonambient PM concentrations generated dependently on personal activities (X_{nap}) which

may correspond to smoking and the personal cloud effect. For a period (T) of constant ambient PM a subject spends time T_a outdoors and time ($T - T_a$) in *n* different nonambient microenvironments. The total exposure to ambient PM can be expressed as:

$$X_{ae} = \frac{[T_a X_a + (T - T_a) X_{na}]}{T} \quad (7\text{-}33)$$

For a nonambient microenvironment, the equilibrium concentration of ambient particles in it will be equal to

$$X_{na} = \frac{X_a P a}{(a + k)} \quad (7\text{-}6)$$

where *P* = penetration fraction of PM in the ambient air entering the nonambient microenvironment,

a = air exchange rate, h^{-1}

k = deposition rate (a function of AD), h^{-1}.

As discussed in section 7.2, the penetration factor *P* is virtually equal to 1 for all particles less than 10 μm (Thatcher and Layton, 1995) and the fraction of X_{na}/X_a is as shown on Figure 7-16. Combining equations 7-33 and 7-6, we obtain

$$X_{ae} = \frac{X_a[T_a + \Sigma t_j a_j/(a_j + k)]}{T} \quad (7\text{-}34)$$

where $T - T_a = \Sigma t_j$, total time spent indoors,

j = 1 to n, index of indoor microenvironment visited.

Defining *z* as the overall ratio of exposure to ambient PM (X_{ae}) to the ambient concentration (X_a), so that $X_{ae} = z X_a$, letting $y = T_a/T$, the fraction of time the subject is outdoors, we obtain the average relation,

$$z = y + (1 - y) \overline{\left(\frac{a}{a + k} \right)}, \quad (7\text{-}35)$$

$$\text{where } \overline{\left(\frac{a}{a+k}\right)} \text{ is a time weighted average.}$$

As shown on Figure 7-38, on a daily basis, *z* can vary by an appreciable amount by spending a fraction (*y*) of time outdoors. For $y = 1/3$ (8 h), exposures to fine ambient $PM_{2.5}$ increase by 100% for people living in homes with an air exchange rate $a = 0.1\ h^{-1}$.

The total exposure (X) can now be written as,

$$X = z\,X_a + \frac{\Sigma\,[(X_{nai})_j + (X_{nap})_j]\,t_j}{T}, \qquad (7\text{-}36)$$

where $\Sigma\,[(X_{nai})_j + (X_{nap})_j]\,t_j / T = \beta$, the personal exposure increment produced by sources that do not influence the ambient concentration as measured by a stationary ambient monitor (SAM). Simplifying, we can rewrite Equation 7-36 as,

$$X = z\,X_a + \beta \qquad (7\text{-}37)$$

which gives a physical significance to the slope and intercepts of the regressions of PEM (X) versus SAM (X_a) as discussed in Section 7.6.

The values of *z*, which depend on *y, a, k* and *P* can be determined from their independent measurements described previously. $P = 1$ for all PM ≤ 10 μm A.D. (Thatcher and Layton, 1995) and $y = 0.074$ [U.S. mean fraction of time spent outdoors per day; U.S. Environmental Protection Agency (1989)]. From PTEAM (Wallace et al., 1993), $a = 0.9\ h^{-1}$ as a median value for night and day. Özkaynak et al. (1993a,b) have determined values for *k* as follows:

For sulfate $k = 0.16\ h^{-1}$

For $PM_{2.5}$ $k = 0.39\ h^{-1}$

For PM_{10} $k = 1.01\ h^{-1}$

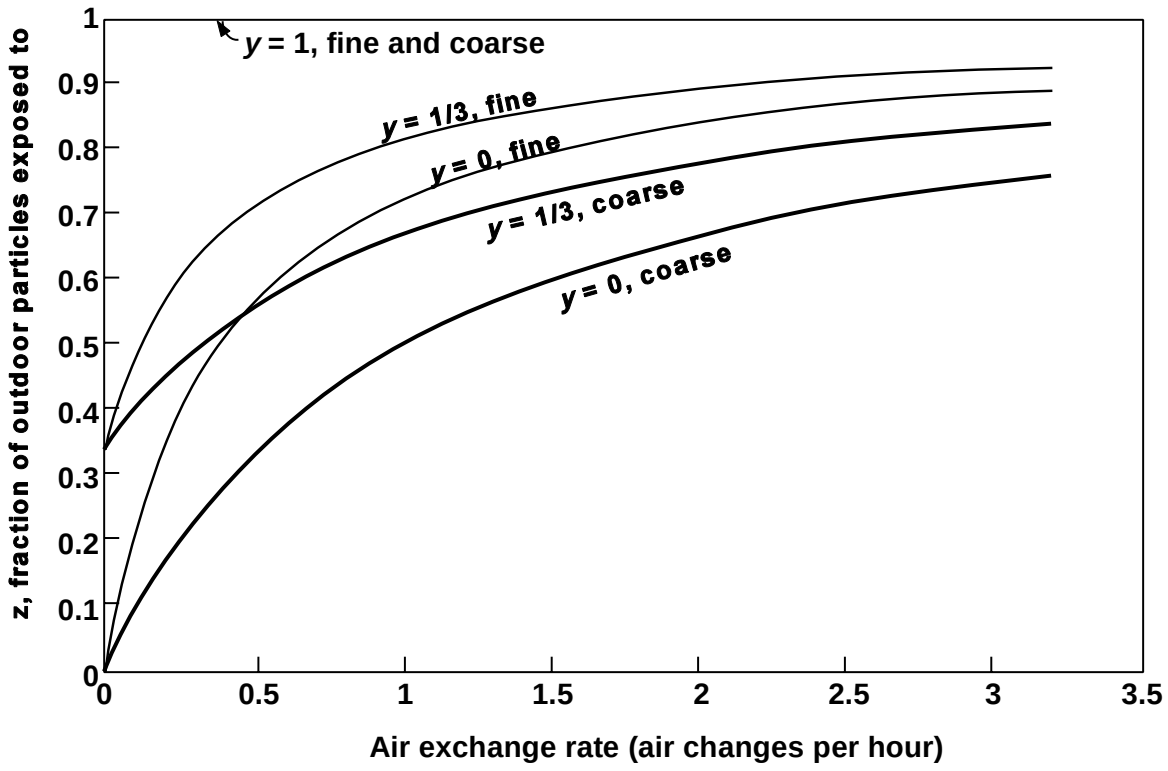

Figure 7-38. Fraction of ambient PM to which people are exposed (*z*) as a function of fraction of time outdoors (*y*) and air exchange rate for fine ($PM_{2.5}$) and coarse (PM_{10} – $PM_{2.5}$) particles.

From the equation $z = y + (1 - y)\, P\, a/(a + k)$

for sulfate, $z = 0.074 + 0.926\ (0.9)/(0.9 + 0.16) = 0.859$

for $PM_{2.5}$ it is $z = 0.074 + 0.926\ (0.9)/(0.9 + 0.39) = 0.720$

for PM_{10} it is $z = 0.074 + 0.926\ (0.9)/(0.9 + 1.01) = 0.512$

These predicted values match closely to the reported values of *z* cited in this Chapter 7 as follows:

Suh et al. (1993) report $z = 0.87 \pm 0.02$ ($r^2 = 0.92$) for $SO_4^=$

Tamura et al. (1996) [Table 7-32] report $z = 0.466$ ($r^2 = 0.905$) for PM_{10},

Lioy et al.(1990) [Table 7-44] report $z = 0.546$ ($r^2 = 0.91$) for PM_{10}

It is not known what the average values of *y* and *a* were for the State College, PA, and Phillipsburg, NJ, cohorts of Suh et al. (1995) and Lioy et al. (1990), or the Tokyo, Japan, cohort of Tamura et al. (1996). Therefore these results can only be considered as tentative at this time.

The parameter ß in Equation 7-37 represents the contribution to personal exposures (PEM) from nonambient sources both independent of and dependent on personal activities. In general the composition of the PM emitted by indoor sources (or resuspended by human activity) that influence ß will be different from the PM emitted into the ambient atmosphere from sources controlled by State Implementation Plans (SIP)s. The nonambient µE emissions are from the activities of the subject (cooking, heating, smoking, resuspension of housedust, hobbies, etc.) or independent activities of others in the same µE that are independent of the ambient concentration (Xa).

For the situation in Tokyo (Tamura and Ando, 1994; Tamura et al., 1996) the PM_{10} PEM vs PM_{10} SAM correlation is good for all subjects individually, as well as their average PEM, because the data were collected in a manner to minimize ß. These data for the seven nonsmoke exposed elderly subjects were culled to remove observations which were influenced by overt particle generating activities such as visitors' smoking, burning of incense, and burning of antimosquito coils. The custom of taking off shoes on entry into Japanese residences and use of "tatami" mat flooring minimized resuspension of PM less than 10 µm AD, although indoor activity did raise dust above 10 µm AD (Tamura et al., 1996).

For the U.S. cities of Phillipsburg, NJ, and Riverside, CA, with large numbers of observations, the correlations of PEM vs SAM for PM_{10} were significantly positive but less than for Tokyo, Japan, possibly due to the passive smoking and house dust generation in the Riverside, CA, and Phillipsburg, NJ, studies. Even so, in Riverside, CA, ambient sources provided about 67% of PM_{10} mass measured indoors (Özkaynak et al., 1996). Finally, the results of the studies in Beijing, China, and Azusa, CA, gave positive correlations of PEM and SAM that were not significantly different from zero (If one outlier is included in the Azusa analysis, the PEM vs SAM correlation is negative). These low correlations may be due to low air exchange rates in Beijing during the winter as evidenced by the low PEM/SAM ratios, and the presence of indoor sources in Azusa, as evidenced by the PEM almost double the SIM or SAM. These latter studies are typical of the results in other U.S. cities such as Kingston and Harriman, TN (Spengler et al., 1985), where ambient pollution is relatively low, so that the personal cloud and indoor source effects predominate.

In summary, it appears that the first exposure conclusion of the previous PM criteria document (U.S. Environmental Protection Agency, 1982), quoted in section 7.1.3, has been

generally supported by recent studies. If the relation of equation 7-35 which appears to predict the observed relations in several studies cited in this document is a reasonable model of the personal exposure to ambient PM, then that conclusion can be adjusted more specifically as follows:

1. Long-term personal exposures to fine PM sulfates of outdoor origin may be estimated by approximately 85% of the sulfate in the fine fraction of ambient PM.

2. Long-term personal exposures to PM < 2.5 μm A.D. of outdoor origin may be estimated by approximately 70% of the PM < 2.5 μm A.D. in the ambient PM.

3. Long-term personal exposures to PM < 10 μm A.D. of outdoor origin may be estimated by approximately 50% of the PM < 10 μm A.D. in the ambient PM.

These relationships still need to be validated in populations other than those from which they were derived. Variations will exist for cohorts with different fractions of time spent outdoors (*y*) and air exchange rates (*a*) than the values chosen for representing the national averages.

Ambient concentrations of PM_{10} measured at properly sited monitoring stations are highly uniform in urban areas (Burton et al., 1996, Suh et al., 1995), have no losses in penetration into μEs (Thatcher and Layton, 1995), and may be highly correlated with personal exposures to PM_{10} (Tamura et al., 1996) where indoor sources of PM_{10} are minimal. Even where indoor sources of PM_{10} exist, they tend to produce different chemical species than those found in the $PM_{2.5}$ fraction, as shown by the sulfates which do not appear in the personal cloud (Özkaynak et al., 1996; Suh et al., 1993).

It is therefore concluded that the presence of variable indoor sources of PM_{10} tends to lower the observed correlations between PEM PM_{10} (derived from both ambient and nonambient sources) and SAM PM_{10} (derived only from ambient sources) and even achieve values nonsignificantly different from zero. Consequently, the use of an ambient concentration of $PM_{2.5}$ or PM_{10} in relation to daily changes of mortality and morbidity may be a reasonable surrogate for the average personal exposure of people in the community to the $PM_{2.5}$ or PM_{10} generated by ambient sources. "The consistently higher R^2 values observed in the longitudinal regressions support the epidemiological findings more strongly than the poor correlations noted in the standard cross-sectional regressions" (Wallace, 1996), as per the U.S. EPA reanalyses shown in Tables 7-36 and 7-42.

7.8 SUMMARY AND CONCLUSIONS

For PM, the total exposure of an individual consists of the summation of the individual's exposure to PM in a variety of microenvironments. This typically includes exposures while (a) outdoors and (b) indoors (at-home or in microenvironments such as shops and public buildings; at-work in an office or factory; and in a vehicle). The principle of superposition is a useful mechanism to visualize the summation process. A simplification of this summation process for an arbitrary individual, described in detail by Figure 7-30, is illustrated in Figure 7-39. In each sub-figure (a to d) of Figure 7-39, the shaded area represents PM exposure (in $\mu g\text{-}h/m^3$) of ambient origin appropriately indexed by a central (community) monitoring station. The clear area represents that PM exposure (in $\mu g\text{-}h/m^3$) the individual is exposed to which is not characterized by the PM measured at the central monitoring station.

Figure 7-39a shows that while outdoors, the subject can be exposed to (a) widely dispersed ambient PM that is represented by the community monitoring station and, independently, also to (b) proximal PM that does not markedly influence the monitoring station reading (from tobacco smoking, standing over a grill at a backyard barbecue, “personal cloud”, etc.). For example, in the PTEAM Study, backyard concentrations of $PM_{2.5}$ and PM_{10} had a correlation on the order of 0.9 with a central monitoring station. Also, in Tokyo (Figure 7-25), outdoor concentrations immediate to the homes of subjects studied by Tamura et al. (1996) had a correlation of 0.9 with the local ambient monitoring station.

Figure 7-39b shows that, while indoors (not at work), the subject can be exposed not only to (a) ambient PM (represented by the monitoring station) that infiltrates indoors but also to (b) PM of indoor origin that does not influence the ambient monitoring station reading (from smoking, cooking, vacuuming, “personal cloud”, etc.). Obviously, the proportion of exposure to PM of ambient origin versus that of indoor origin can vary widely, depending on: outdoor concentrations of the ambient PM; the air exchange rate of indoor spaces; the

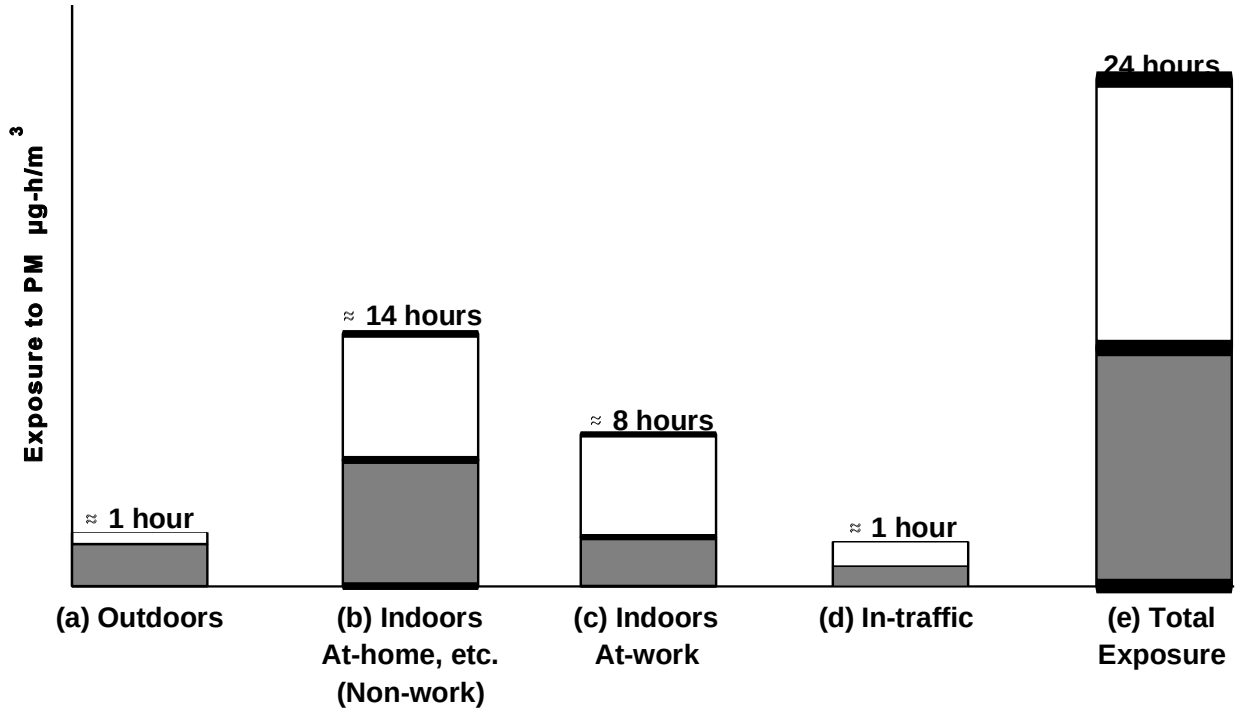

Figure 7-39. Conceptual representation of potential contributions of PM of ambient origin and PM generated indoors to total human exposure of a hypothetical individual. The total personal exposure (e) of that individual will consist of the sum of exposures to widely dispersed PM of ambient origin (shaded areas) characterized by measurements at a centrally-located community monitoring site and all other exposures (non-shaded areas) to proximally generated particles either outdoors or indoors in situations designated for (a), (b), (c), and (d). Times of exposure in the various situations reflect typical time-action patterns for U.S. adults. Depicted exposures to PM of non-ambient origin may vary greatly from those shown there for qualitative impression only, depending on various factors described in the text.

presence or absence of indoor PM sources; and the removal efficiency of indoor sinks for specific constituents of the respective PM of ambient or indoor origin. In the absence of major indoor PM sources (e.g., smoking), the percentage of total exposure contributed by PM of ambient origin can be substantial. For example, as shown in Table 7-2, between 60% and 80% of indoor air PM was estimated by source apportionment methods to be of ambient origin in non-smokers' homes in two U.S. cities (Steubenville, OH; Portage, WI) included in the Harvard Six-City Study. Even in smokers' homes, it was estimated that 60% of the non-smoking related PM was of ambient origin in the same two cities. The New York State ERDA Study (see page

7-23) also showed that, in homes without combustion sources, 60% of the total indoor $PM_{2.5}$ was from outdoor sources. For homes with smokers in the same study, about 66% of the non-tobacco smoke indoor particles were found to be of ambient origin. Similarly, based on the Tamura et al. (1996) data shown in Figure 7-24, it can be estimated that as much as 80% of the measured indoor PM_{10} in Japanese homes without combustion sources was of ambient origin.

Figure 7-39c shows that while indoors at work the subject can also be exposed to (a) ambient PM (represented by the community monitoring station) which infiltrates indoors, and (b) PM of indoor origin that does not influence the monitoring station reading (from smoking, welding, machining, "personal cloud", etc.). It can be expected that, for office-type work, similar relationships as described above for the other indoor conditions (e.g., smokers' or non-smokers' homes) would apply. However, for work conditions involving particle generation (e.g., wood working, welding, mining, etc.), the personal exposure of "dusty-trade" workers to indoor-generated particles can be several orders of magnitude greater than their exposure to indoor particles of ambient origin.

Figure 7-39d shows that while in traffic, the subject can be exposed to (a) ambient PM that is represented by the monitoring station (via ambient air infiltration into the vehicle), and (b) PM of on-board or proximal vehicle origin that does not directly influence the community monitoring station reading (from smoking, exhaust penetration from nearby vehicles, etc.). For example, in one study, Morandi et al. (1988) found that the average concentration of $PM_{3.5}$ in motor vehicles in traffic (55 $\mu g/m^3$) was 60% higher than the average outdoor $PM_{3.5}$ level (35 $\mu g/m^3$).

Figure 7-39e is a simple rearrangement of the shaded and non-shaded areas to show that an individual's total daily exposure ($\mu g\text{-}h/m^3$) can be thought of as the sum of two quantities: (a) exposure to PM characterized by the local community monitoring station, and (b) exposure to PM of immediately proximal origin that varies independently of the PM measured at the monitoring station. Conceptually, everyone in the community will be exposed to the mix of PM represented by the shaded area that is characterized by the local monitoring station, due to their time outdoors and the penetration of PM into indoor microenvironments and vehicles. However, not everyone in the community will be exposed to the identical mix of PM represented by the clear area, because this exposure and its chemical composition is idiosyncratically related to their individual habits and practices (smoking, home cleanliness, hobbies, "personal cloud", etc.),

their occupation (home maker, student, office worker, welder, miner, etc.) and their mode and usage of transportation (car, bus, train, etc.).

Evaluation of information useful in determining relative contributions of ambient (outdoor) and non-ambient (indoor) particles to total human exposures leads to the following key conclusions:

(1) For PM, the ambient environment can be a major source of indoor pollution due to air exchange and infiltration. Whether the ambient is the dominant source of indoor PM depends on the relative magnitude of indoor sources of PM.

(2) For PM of size fractions that include coarse particles, some studies have identified statistically significant relationships between personal exposures and ambient concentrations, while other studies have not, probably due to overwhelming effects of indoor sources, "personal clouds" and other individual activities.

(3) Cross-sectional regressions of personal exposure on outdoor $PM_{2.5}$ and PM_{10} concentrations generally explain less than 25% of the variance ($R^2 < 0.25$). However, longitudinal regressions for each person in the study (in those cases where the person was measured repeatedly) often show much better relationships between personal exposure and outdoor air concentrations.

(4) Personal exposures to outdoor-generated PM of any size fraction $\leq PM_{10}$ can be estimated from the fraction of time spent indoors and an estimate of the air exchange rate and deposition rate associated with that size fraction.

(5) The relationship between ambient concentration and personal exposure is better for finer size fractions of ambient PM, than for coarser PM. Higher correlations between ambient concentration and personal exposures have been found for fine PM constituents (such as sulfates) without indoor sources.

(6) For a study population of nonsmokers in which there is a significant positive correlation between personal exposures and ambient concentrations, the ambient concentration can predict the mean personal exposure with much less uncertainty than it can predict the personal exposure of any given individual.

(7) For Riverside, CA, where 25% of the nonsmoking population was estimated to have personal exposures on the day they were monitored that exceeded the 24-h National Ambient Air Quality Standard for PM_{10} of 150 $\mu g/m^3$, approximately 50% of this mass was found to be of ambient origin.

(8) The personal exposure to PM of smokers is dominated by the milligram quantities of PM inhaled with each cigar, pipe, or cigarette smoked.

(9) For the U.S. studies, almost all personal exposures to PM are greater than the ambient concentrations.

(10) The penetration factor from outdoors to indoors for both $PM_{2.5}$ and PM_{10} was found to be unity in the PTEAM and Thatcher and Layton (1995) studies.

(11) Deposition rates in indoor microenvironments for PM_{10} and its fine and coarse fractions were determined in the PTEAM Study. Similar deposition rates were found by Thatcher and Layton (1995). Deposition reduces exposure to ambient PM; coarse mode PM is removed more rapidly than $PM_{2.5}$, which is removed more rapidly than sulfate.

(12) Under equilibrium conditions, residential indoor concentrations of outdoor-generated PM of any size fraction $\leq PM_{10}$ can be estimated for any given air exchange rate, by employing the deposition rate associated with that size fraction.

(13) For PM, studies have detected a "personal cloud" related to the activities of an individual who may generate significant levels of airborne PM in his/her vicinity which may not be picked up by an indoor PM monitor at a distance.

(14) There is some evidence that nonsmoke-exposed elderly people have lower residential indoor PM concentrations than the simultaneous ambient PM concentrations, as opposed to the general population who have indoor PM concentrations comparable to or greater than ambient PM concentrations.

(15) Measured indoor air concentrations of $PM_{2.5}$ and PM_{10} generally exceed outdoor air concentrations (often by a factor of two) except in areas where outdoor concentrations are high (e.g., Steubenville, OH and Riverside, CA).

(16) Indoor concentrations are higher during the day than at night.

(17) Correlations between indoor and outdoor particle mass concentrations were not significant in two of the three major studies reviewed. In the third (PTEAM) study, they ranged between 0.22 and 0.54.

(18) Regressions of indoor on outdoor $PM_{2.5}$ and PM_{10} concentrations generally explain less than half of the variance ($R^2 < 50\%$) if the regressions are carried out simultaneously on all homes in the study. However, regressions for a single home (in those cases where homes were measured repeatedly) often have much better indoor-outdoor relationships (R^2 up to 90%). Since most epidemiological studies deal with repeated measurements over time, "longitudinal" regressions by individual home may be more relevant to these studies than "cross-sectional" regressions across all homes.

(19) The largest identified indoor source of particles in both homes and buildings is cigarette smoking. Homes with smokers have an ETS-related $PM_{2.5}$ concentration increment ranging between 25 and 45 $\mu g/m^3$.

(20) The second largest identified indoor source of particles is cooking. Homes with cooking had increased levels of PM_{10} on the order of 10 to 20 $\mu g/m^3$.

(21) Unknown indoor sources accounted for a substantial fraction (25%) of indoor concentrations of both $PM_{2.5}$ and PM_{10} in the PTEAM Study. These sources appear to be due to personal activities, including resuspension of house dust.

(22) Variations in personal exposure due to fluctuations produced by indoor sources of PM are independent of the variations in personal exposure produced by ambient sources.